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ARCHIVES OF OPHTHALMOLOGY.

# ON THE DECLINATIONS OF THE VERTICAL MERIDIANS OF THE RETINA.

BY GEORGE T. STEVENS, M.D., PH.D., NEW YORK.

*(With nine line cuts in the text.)*

*Third Paper.*

HAVING examined to some extent the nature, characteristics, and method of detecting and measuring the declination, at least so far as it is manifest, we are in position to inquire whether the various forms of declination are sources of inconvenience, discomfort, or distress to the individual subject to either form of the anomalies.

As I have insisted upon the direct relation between declination and heterophoria or heterotropia, the former being the causative element of the two latter, it follows (if this view is correct, and the view has been put to test in many hundreds of cases and the assertion of causative relation has been confirmed beyond doubt) that the evil influences of strabismus and of heterophoria are the more immediate and apparent influences of declination. More than this, there may be a condition of declination which acts as a direct source of great nervous trouble, of asthenopia or of amblyopia, while no marked heterophoria may be manifest. Hence the disadvantages of declination are by no means confined to the induction of heterophoria.

To speak of strabismus first, its injurious effects upon vision and its disfiguring effect upon the facial expression are sufficient to cause it to be regarded as a baneful defect, injurious to the health, to the prospects, and often to the morals of the victim. It is hardly necessary to discuss this form of the result of declination further; its hurtful features are too evident to require extended notice in this immediate connection.

As to the evils resulting from conditions of heterophoria, we know some of them though the disadvantages of these anomalies have been by far too lightly regarded even by ophthalmologists.

Headaches, chorea, asthenopia, a long list of disturbances of the functions of the nervous system, these are among the pernicious influences of heterophoria, but we may pass at once to the more direct hurtful tendencies of declination.

I have spoken of the influence of declination upon the expression of the face. This is by no means as light a matter as it might be supposed, indeed, it is an extremely important matter. A very large proportion of constantly recurring headaches, cases of which are found in every walk of life and in great numbers in each, are the immediate effect of the habitual tension of certain of the facial muscles which are engaged in adjusting the eyes to overcome some form of declination. It is a great mistake to suppose that the muscles within the orbits are the only ones responsible for giving the eyes their proper relations to each other, to the cranium of the individual, and to the objects to which they are directed.

Not only the muscles within the orbits and those in the immediate vicinity of these cavities but muscles at a considerable distance, as at the back of the head, and even muscles controlling the pose of the body, are directly or indirectly brought into the service of the adjustments of the eyes.

I have considered the statement just made. It is not a careless statement, it is not absurd or extreme, it is not even too emphatic. It understates the facts.

Naturally when we consider the great number of forces which may be brought into requisition and the large number which are in many cases habitually brought into service we may well conclude that a disarrangement of the adjustments of the eyes may induce a great variety of unpleasant effects.

Space does not permit any detailed review of these undesirable effects but we may recall a few of them in passing.

Much has been written of vertigo and of the influences of certain parts of the auditory apparatus in its induction. As a matter of fact, for every case of vertigo arising from such an origin, scores arise as the effects of declination.

So also we may say of a large number of disorders of the nervous centers, including some of the most important, that while certain forms evidently depend upon ascertainable lesions, for example, mechanical pressure, tumors, manifest degenerations, and other forms of evident physical disturbance, a larger proportion are not necessarily dependent upon such organic lesions. Of these last a notable proportion are directly or indirectly related to peculiarities in the adjustments of the eyes.

Owing, in some degree, to an absence of knowledge, but perhaps more frequently to a conservatism in preserving familiar terms to describe certain physical manifestations having some resemblance, we prefer to be misled by calling by the same name affections of an absolutely different nature and arising from causes as unlike as possible. For example, we speak of a form of convulsive attacks, for which we have found no pathology, as epilepsy. Another form of convulsive crisis, quite different, and which is evidently the result of pressure upon the great nerve centers, we also call epileptic attacks. The absurdity of such a classification is too evident to discuss.

Now, it is true that a very important proportion of the disturbances to the physiological performances of the nervous centers are the direct or indirect results of unfavorable adjustments of the eyes. I am too well aware that such a statement will be received by many with incredulity. Incredulity, however, does not modify the facts.

Of many of the more indirect forms of nervous affections arising from the pose of the body as influenced by the adjustments of the eyes I have spoken in a series of articles published in the *New York Medical Journal* of 1910 and 1911. It is not my purpose to discuss that subject here at any length and the reason for referring at all to the general ill effects which may arise from unfavorable adjustments of the eyes is only to

impress the truth of the importance of the subject which we are considering.

We may now turn our attention to methods for correcting or for amelioration of the anomalies of declination.

The first question that would naturally occur to us is: Can we correct the declination or favorably modify it by glasses?

That a correction by lenses would be impossible would follow when we consider that the defect is not one of refraction, of weakness, "insufficiency," or of development of the adjusting muscles of the eyes, but one of position of the globe of the eye in the orbit. However, in certain exceptional cases, lenses, if they have considerable refractive influence, may serve as a more or less useful modification of the conditions. This is shown when strong convex lenses are placed before strabismic eyes. The squint, in a certain proportion of cases, partly (never wholly) disappears when such glasses are used. This was long ago attributed to the influence of the lenses on the accommodation. Many years ago I showed that this view is entirely incorrect. It would demand too much space here to traverse this subject but I shall content myself by saying, as I have before, that the eyes *find* certain points in the glasses where, if the glasses are of high refracting grade, the image will be subjected to a certain amount of deflection which will, in proportion to this effect, remove the sense of tilting of the image as seen by the unaided eye. This enables the eyes, so long as this effect continues, to assume somewhat more normal relations, but these relations are no longer maintained when the glass is removed and of course the squint returns. In other conditions also such strong lenses may serve to modify the effects of declination but, of course, they can never *correct* the leanings of the meridians. The effect of such a modification is, therefore, temporary, imperfect, and only moderately useful and only occurs when very strong lenses are used.

When we come to the question of an actual correction we must of necessity come to a surgical operation.

The logical operation would be a readjustment of the orbit, but since that is quite out of the question we have to use the conditions at our disposal, which means that we must modify the insertion of the muscles that control the eye in such a way as to place the retinal meridians in the most favorable position.

This is not the work of an apprentice. It is a proceeding demanding the best judgment and the highest operative skill of the surgeon.

To accomplish a rational and proper correction of declination certain indispensable conditions are required.

First, *the normal rotation ability of no ocular muscle should be restricted.* This is a most important condition.

Many years ago when I was accustomed to practice contractions of tendons or graduated tenotomies in cases of heterophoria, I observed and in my writings impressed the necessity of great care to maintain as far as possible the full rotating power of the muscles involved in the operation, directly or indirectly. But notwithstanding this care, which was scrupulously observed in my own work, my purpose of maintaining a full rotative effect of the directing muscles was far too often thwarted. In the practice of those who give no consideration to this feature of course it is a routine result that the rotating effect of one or more muscles is impaired; indeed it is a daily occurrence to see someone for whom the rotation ability of one or more eye muscle has been almost or completely sacrificed. To say that such a result is a misfortune is to state the fact in the mildest form.

In this journal,[1] and elsewhere, long before the observation of the relation between heterophoric conditions and declination, I had expressed the hope and the view that we would sometime be able to dispense with all shortening and all setting back of any eye muscle in the correction of heterophoria.

Although these remarks are somewhat aside from our discussion, I am constrained to introduce them here because, with the knowledge of the connection between heterophoria and declination the necessity for any reduction of the action of any eye muscle disappeared and I cannot too forcibly urge that settings back or advancements of the insertion of ocular muscles be universally abandoned as means of correcting either strabismus or heterophoria. Personally I have with less than three exceptions not practiced either graduated tenotomy or tendon shortening for any eye muscle during the past fifteen years, yet I have operated for many hundreds of cases of heterophoria and strabismus. I must add to what I have said

[1] ARCHIVES OF OPHTHALMOLOGY, 1889, p. 378, etc.

in this connection *an earnest protest against performing any operation for disabling any eye muscle of a sound eye because a muscle of the other eye is disabled either by operation or by paralysis.* Such an operation would be comparable to amputating a leg because its fellow is too short. *Any operation to restrict or injure the action of a sound muscle of a sound eye in order to compensate for a defect or a supposed defect of the musculature of the fellow eye is an opprobrium to surgery for which there can be no possible excuse under any imaginable circumstances.*

Returning from our digression, let us repeat with emphasis that the rotation effect of an eye muscle should never, in operating for heterophoria, strabismus, or declination, be restricted or injured.

Next, the conjunctiva, although a membrane which usually heals quickly even from a somewhat extensive wound, should not be opened except to the extent of absolute necessity. In the usual operations for declination (which means for heterophoria and strabismus also) the wound need not be more than one millimeter in extent.

### OPERATIVE MEASURES FOR DECLINATION.

Many years ago I suggested several methods for operative correction of declinations, but, as a result of experience, that which I shall here describe has been found in all respects so superior to the others that I shall confine myself to describing what I have called the operation for

### EXTENDO-CONTRACTION.

Inconsistent as the term may at first appear it accurately describes the effect of the procedure as will be seen as we proceed. By this operation the full rotation action of the muscle is preserved, heterophoria is not induced, eyes which were adjusted in parallelism before the operation continue to be so adjusted after it, while by means of the operation a very considerable degree of the leaning of the meridians can be removed. This does not mean that by any single operation a high degree of declination may be expected to yield; the

leverage is limited by the width of the tendon on which the operation is performed. The tendons are not alike in their attachment to the eyeball, some having a broader attachment than others. In this respect the insertion of the internal rectus not only affords a broader attachment than any of the other recti but for other reasons offers greater advantages as a point of influence than any other tendon of the straight muscles.


FIG. 10.—Flexible Eye Speculum.

Let it be supposed, then, that we are proposing to do an operation for extendo-contraction at the insertion of the internal rectus tendon of the left eye. What are the procedures?

I shall, in answer to this question, use, with slight modifications, the language in which I described the operation in 1901,[1] repeating what was then said, that it is of the highest importance that the various steps should be taken exactly in the order and in the manner described.

FIG. 11.—Lid Retractor.

Assuming that we are to operate at the insertion of the left internus, and for a positive (+) declination, the surgeon will stand first at the right-hand side of his patient, whose eyelids are separated by a speculum (Fig. 10) which is more flexible and yet in all these cases more serviceable than most other forms. The patient is directed to turn the eyes to the left and downward. (If the operation is to be done on a superior rectus the lid retractor, Fig. 11, is used.)

Then, the surgeon, seizing a very small fold of the conjunc-

[1] *New York Medical Journal*, February, 1901.

tiva with the fine forceps (Fig. 12), snips a transverse opening through the membrane with his fine-pointed scissors (Fig. 13) just over the upper border of the insertion of the tendon and about one millimeter in extent and, pushing the blades of the scissors forward while avoiding, as far as possible, enlargement


FIG. 12.—Fine Forceps.

of the small opening of the conjunctiva, which will stretch to some extent without forming any permanent extension of the wound, a pocket is to be formed reaching nearly to the border of the cornea and extending downward to the lower border of the tendon insertion and upward a few millimeters above the


FIG. 13.—Scissors.

upper border of the cornea. Of course the surgeon does not see the tendon but judges of the extent of the pocket by his knowledge of the anatomy of the parts.

The pocket having been formed, the operator next passes to the left side of the patient. He now seizes the insertion of the tendon at its upper border in the fine blades of the forceps, and


FIG. 14.—Fine Sharp Hook.

separates it from the sclera by very careful snips to the extent of two or three millimeters only. He then introduces the delicate sharp hook (Fig. 14) between this separated part of the tendon and the sclera and, when it has been carried back to the desired extent, presses the sharp point against the inner surface of the tendon and draws the latter forward. The hook should engage itself sufficiently below the upper border of the

tendon and far enough back to insure the drawing forward of that part of the tendon.

This small portion of tendon is now forced through the little opening in the conjunctiva, which will stretch to a considerable extent. Now a small curved needle held by a suitable needle-holder (Fig. 15), one of two needles attached to a very

FIG. 15.—Needle-holder.

fine but strong thread of silk, is carried through the protruding portion of the tendon which is then allowed to retreat within the conjunctiva. An assistant takes the two needles, holding the thread out of the way of the operator, who now, using the small tendon hook (Fig. 16), separates by the fine points of


FIG. 16.—Small Tendon Hook.

his scissors the remainder of the tendon insertion from the surface of the sclera with much care not to enlarge the original conjunctival wound. If the tendon is unusually broad a counter opening can be made near the lower border of the tendon for the fixation of a second suture. This will be very rarely, if ever, required. The extent of the division can be felt by the operator with his small tendon hook, when, if satisfactory, the next step of the operation can be entered upon.

An assistant now inserts a director (Fig. 17) through the

FIG. 17.—Grooved Director.

conjunctival wound into the pocket, carrying it somewhat above the border of the cornea. One needle is carried in the direction of the probe, making its exit above and near the cornea. The second needle is then carried through at three or four millimeters below and the thread is drawn and tied. Until the surgeon has become familiar with the process it will

be best first to make a slip knot, then test the effect of the traction by the phorometer before making the knot fast. An allowance of about 10° can be made for the modification of tension as the process of union proceeds. The patient, should there be found a sufficient degree of excess of tension, may be permitted to use the eyes with freedom, but should the allowance not be sufficient the eyes should be kept for the greater part of the time, until union occurs, closed in order that as little as possible be lost of the tension.

Eight days after the operation, sooner if there is esophoria

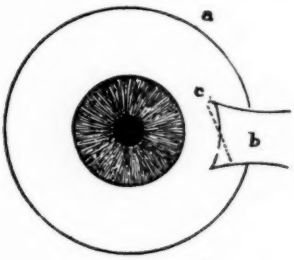


FIG. 18.

which would probably remain if the support should continue to exert too great traction, the suture is to be removed.

The accompanying diagram (Fig. 18) will suggest what is expected to be accomplished by the operation. The equator of the eye is indicated by the line *a* and the tendon of the internal rectus by *b*. The solid line represents the position and direction of the insertion of the tendon before the operation, while the dotted line suggests the position of the insertion when healing has occurred.

It will be seen that while the upper extremity of the tendon at *c* has been advanced toward the cornea, the lower extremity has fallen back, giving the insertion an oblique direction. The result of this oblique direction of the insertion will be to influence the vertical meridian to lean toward the medial plane, while the rotation of the eye outward or inward should not be in any degree restricted.

As a matter of experience it is found that in cases where declination of a considerable degree has been thus corrected

the rotations in all directions are somewhat improved, indeed, in cases of correction of a very high degree the rotation in all directions may be greatly improved.

I have confined the description of the operation to the tendon of the internal rectus in the interest of clearness. Of course an operation on the externus, observing the principle, can be done with the modification of advancing the lower angle of the tendon, and on the superior rectus by a corresponding advancing of the external angle. On the other hand, for negative declination the opposite angle is in each case to be chosen.

While we may choose, for convenience, either the internus the externus, or the superior rectus for an extendo-contraction, there is urgent reason why the tendon of the inferior rectus should never be subjected to operative interference. In the order here stated the most favorable results may be obtained; the internus, the externus, the superior. As a mechanical fact we can produce a greater effect upon the superior than upon the external rectus. The advantage of the external rectus is that there is less risk of inducing exophoria by an operation on the externus than on the superior, contradictory as this may appear.

In regard to the selection of the tendon for operation, the operator may also consider the condition of heterophoria, if present. Thus, for example, should a pronounced degree of esophoria be present he might, because of the immediate effect, prefer to operate on the externus. This, however, is not essential. It is my custom in cases of converging strabismus to operate either upon the external or internal rectus rather indifferently. The immediate effect might be greater from an operation on the externus. The main, indeed, the only point is to reduce the declination while preserving absolutely all the rotations.

We find in a very large proportion of cases a positive declination of one eye and a negative of the other. It is very important to proceed with care in these cases. Theoretically it is possible to find a case of negative declination which is entirely independent of a positive declination of the fellow eye or even negative declination of both eyes. I have never seen a case of one or the other form. In a very considerable proportion of

cases the negative declination as shown by the clinoscope is the result of synergic tension in the effort to resist the tendency to the tilt of the eye with the positive declination. It often happens that with the correction of the positive form of declination the negative form disappears. It is therefore important to wait for a correction or at least an approximate correction of the positive form before any attempt is made to correct the other.

The foregoing suggestions in regard to the declination which *always* exists as a cause of heterophoria should serve us as a safe guide in the treatment of the lesser forms of deviation or of deviating tendencies of the axes of the eyes. When we reach the higher degrees of strabismus we are sometimes left without very obvious indications of the underlying fault. The patient may be unable to see one or other of the indicators of the clinoscope at all or only one at a time. In some cases he will insist that he sees both but it is evident that he does not see them simultaneously. Hence he will apparently have a more complete absence of declination than the most favorably adjusted eyes. In these last cases much patient care may at length reveal the truth. In those in which no valuable tests can be obtained either by the clinoscope or lens clinoscope we should, as I have suggested above, resort to observation of the movements of the eye from exclusion to fixation, when a *moderate* tentative operation may be performed. This will probably lead to more satisfactory tests after which the treatment may be proceeded with in the ordinary manner.

The rational method of treatment of strabismus is much less expeditious than the old method of destroying the rotation ability of a muscle or shortening another muscle so as to haul an eye into apparent position but it is the duty of a surgeon to get the best, not the quickest, results, and many weeks spent in correcting a strabismus in such a way as to leave every movement free is infinitely to be preferred to a quick severance of a muscle with only an extremely unsatisfactory cosmetic result as the outcome.

The aim of every strabismus operation should be perfect and easy binocular vision, which, by the methods formerly practiced, was never, notwithstanding many claims unsupported by facts, in any case obtained.

# PROGRESSIVE MACULAR DEGENERATION IN THREE MEMBERS OF A FAMILY.[1]

By MARCUS FEINGOLD, M.D., New Orleans, La.

*(With five illustrations in color on Text-Plates XXVI and XXVII.)*

WITH the report in 1897 by R. D. Batten of the first instance of "symmetrical disease of the macula" in two brothers, a new clinical unit was created which, once established, was soon found and recognized on all sides. And so it came that in spite of the short time since that date ophthalmic literature boasts now of a number of case reports and of at least two thorough treatises on the subject. But in spite of this a good many points remain to be cleared up and for that reason I believe the report of the three following cases will be excused.

The three patients are children of Russian Jews who are first cousins. Both parents, the only offspring of the father's first marriage, and eight of the nine living children were examined. Nos. 1, 2, and 6 are the patients under consideration and the results of their examination will be given separately.

*Mr. Seb. P.*, aged 66.
V. O. D. $\frac{5}{20}$; V. O. S. $\frac{5}{10}$?. V. O. D. + 2.5s $\frac{5}{4}$; V. O. S. + 2.5s$\frac{5}{4}$.
Retinoscopy under homatropin: Right and Left + 2.5s.
Ophthalmoscopy: Irregular crescent of diminished retinal pigment at temporal margin of disks; cilio-retinal artery in temporal margin of left disk.
*Mrs. Seb. P.*, aged 59.
Sees better in the dark.
V. O. D. $\frac{5}{7.5}$?; V. O. S. $\frac{5}{7.5}$?

[1] Read in part at meeting of A. M. A., Detroit, 1916.

Retinoscopy under homatropin: R. + 0.5s. to + 0.5 axis 180°.

Retinoscopy under homatropin: L. + 0.5s. to + 0.5 axis 180°.

Ophthalmoscopy: Well-defined crescent of lessened retinal pigment at temporal margin of disks, ½ disk diameter wide in right eye, ⅓ disk diameter in left eye. Irregular frill-like opacity around posterior pole of each lens, about 4*mm* in diameter (+ 18 D. in ophthalmoscope) and more dense in right eye, about 3*mm* and thinner in left eye.

*Louis P.*, aged 45. (Son of first marriage.)

Only ophthalmoscopic examination without homatropin could be made.

Normal, healthy fundus each eye.

1. *Mrs. Harry D.*, aged 38.
   See details below.
2. *Mrs. Ben B.*, aged 36.
   See details below.
3. A girl, died when 18 months old.
4. *David P.*, aged 32.
   Color-blind.
   V. O. D. $\frac{5}{6}$?; V. O. S. $\frac{5}{6}$?
   Retinoscopy under homatropin: R. + 3.5s ⊂ + 1.0 axis 75°; L. + 3.5s ⊂ + 2.5 axis 105°.
   Ophthalmoscopy: Small lump black of pigment at upper temporal margin of left macula.
5. *Maurice P.*, aged 28.
   Color-blind.
   V. O. D. $\frac{5}{9}$?; V. O. S. $\frac{5}{9}$?.
   Retinoscopy under homatropin: R. + 1.25s; L. + 1.5s.
   Ophthalmoscopy: Each fundus normal, healthy. Small vesicle in anterior cortex of right lens; small, round opacity as posterior pole of each lens; small vesicle near posterior pole of left lens.
6. *Herbert P.*, aged 26.
   See details below.
7. *Henry P.*, aged 24. Not living in Louisiana, not examined.
8. *Sam P.*, aged 20.
   Color-blind; horizontal diameter of each cornea, 11*mm*.
   V. O. D. $\frac{5}{9}$; V. O. S. $\frac{5}{9}$.
   Retinoscopy under homatropin: R. + 0.5s.; L. + 0.5s.
   Ophthalmoscopy: Normal, healthy fundus each eye.
9. *Irbin P.*, aged 19.
   Color-blind.
   V. O. D. $\frac{5}{9}$; V. O. S. $\frac{5}{9}$.
   Ophthalmoscopy: Normal, healthy fundus; cilio-retinal

ILLUSTRATING DR. FEINGOLD'S ARTICLE ON "PROGRESSIVE MACULAR DEGENERATION IN THREE MEMBERS OF A FAMILY."

FIG. 1.

FIG. 2.

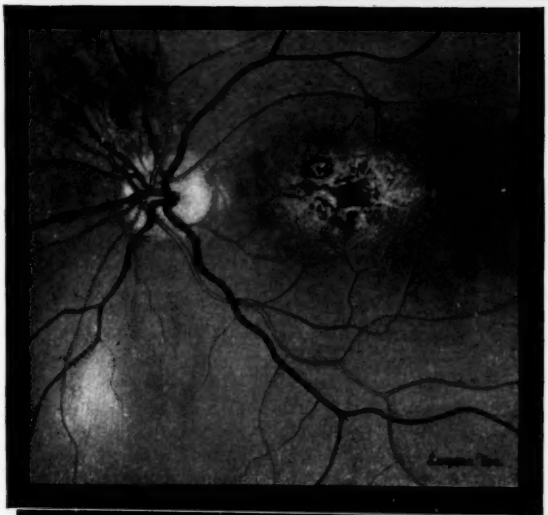

FIG. 3.

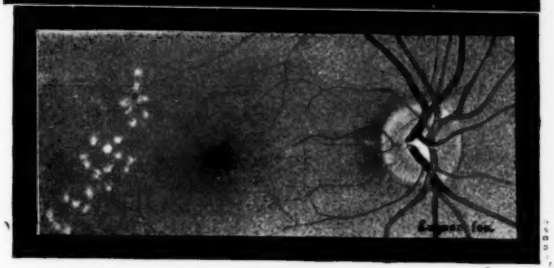

FIG. 1.—Right eye, Mrs. D. Typical focus of degeneration in the macula; focus chorio-retinitis at nasal side of disk. Most advanced stage.

FIG. 2.—Left eye, Mrs. D. Typical focus of degeneration in the macula; focus of chorio-retinitis at upper nasal side of disk. Fresh focus (?) below disk. Most advanced and progressive condition.

FIG. 3.—Right eye, Mrs. B., Case 2. The purplish-red round spot in the macula with absence of foveal reflex represents the first stage of the disease. (Colloid bodies to temporal side of macula.)

artery in temporal margin of each disk, very large in right eye.

10. *Fannie P.*, aged 16.
V. O. D. $\frac{5}{5}$; V. O. S. $\frac{5}{5}$.
Retinoscopy under homatropin: R. +1.5s ⊂ + 0.5 axis 90°; L. + 3.0s ⊂ + 0.5 axis 90°.
Ophthalmoscopy: Line of slightly irregular pigment around disks.

CASE 1.—Mrs. Harry D., aged 38, was first examined during August, 1907, for some difficulty at near work. The extensive macular changes while unusual and attracting attention did not then stand out sufficiently as something extraordinary and singular because of the accompanying extensive retinochoroidal changes in other places of each fundus. Only when her brother, Herbert P. (No. 6), some five years later called for indefinite complaints of blurred vision did the singular appearance of his macular region suggest the idea of some inherited or family disease. This thought led to the examination of the whole family when Mrs. D. was again studied in detail and the changes in Mrs. B.'s fundus were discovered.

V. O. D. $\frac{5}{6}$?; V. O. S. $\frac{5}{6}$?.

Retinoscopy under homatropin: R. + 1.0s ⊂ + 0.5 axis 90°; L. ditto; with this correction: V.O.D. $\frac{5}{6}$?; V.O.S. $\frac{5}{6}$?.

*Ophthalmoscopic Examination:*[1] R. E. Disk round, fairly well defined, not elevated; small shallow physiological excavation; slightly irregular blackish pigmentation at nasal margin. Beginning at nasal margin of disk, an irregular, not well-defined area, slightly larger than disk, in which apparently absorption of retinal and choroidal pigment; here choroidal vessels more plainly visible and some accumulation of black pigment in lumps; area is bounded above by vessels going to the nasal fundus. At temporal side of disk, between it and the macula, atrophy of retinal pigment makes choroidal vessels more clear and allows the intervascular spaces of choroid to stand out more deeply pigmented.

For the purpose of the description of the macular region it will be best to distinguish two areas: an outer and a central or foveolar area. The outer macular area forms an irregular, horizontal rhomboid, with outlines not sharply defined and a horizontal diameter of over two disk diameters; this area is characterized by a grayish-yellow color

[1] The difference in the magnification of the illustrations is due to the manner by which the pictures were painted. In Cases 1 and 2 the fundus was viewed by the indirect method of the Gullstrand, while in Case 3, by direct ophthalmoscopy.

with a veil of very lively retinal reflexes in front. The color is due to very marked pigment absorption, and in this area is found pigment in irregular lumps, fine lines, and dots. The central area, or the area around the foveola (Dimmer), corresponds in shape more or less to the outer one, lies nearer to its nasal margin, and is ½ disk diameter wide in horizontal direction; it is fairly well defined, bluish- or purplish-red in color, and more saturated in the center than at the periphery.

With the binocular Gullstrand one can see distinctly that the pigment in the macular area lies distinctly in the innermost layers of the retina, that the yellowish, silky appearance of the outer macular area is due to an irregularly mottled appearance of the outer layers of the retina—possibly to advanced pigment absorption of retina and choroid which allows the sclera to show through. The bluish-red, more central foveal area seems to derive its color and appearance from conditions lying farther back than innermost layers of retina and again farther back than the surrounding yellowish area.

Balance of fundus and media, normal, healthy.

L. E. Disk round, fairly well defined, not elevated; small physiological excavation. Small area of pigment absorption near temporal margin of disk. At upper nasal margin of the disk a similar area as at nasal margin of the right disk; area fairly well defined, somewhat larger than disk; marked atrophy of retinal pigment allows choroidal details to be seen clearly. Between disk and macular area changes as in right eye, but much less marked. The changes in the macular region are almost identical with those of the right eye but for slight differences in the form of the general contour of the outer macular area, and in the appearance of the pigment spots that are on the whole much finer in the left eye. The central area is again only slightly different in form, but its bluish-red color is more uniform and more saturated than in the right eye. About one disk diameter below the disk, along a vein running almost vertically down, is seen an area somewhat smaller than the disk, its margins gradually fading into surrounding retina; area is yellowish-white in color, shows markedly regular, finely striped appearance which reminds one of medullated nerve fibers; the whole lying distinctly behind retinal vessels and is possibly somewhat elevated: fresh focus? Balance of fundus normal, healthy; media clear.

Field examination of each eye with a round disk of 5*mm* diameter showed almost normal field for white and only slightly restricted field for blue and red; no scotomata could be elicited anywhere. (This examination on the whole

ILLUSTRATING DR. FEINGOLD'S ARTICLE ON "PROGRESSIVE MACULAR DEGENERATION IN THREE MEMBERS OF A FAMILY."

FIG. 4.—Left eye, Mrs. B., Case 2. Description as in Fig. 3.

FIG. 5.—Right eye, Mr. P., Case 3. Typical focus in macula consisting of central bluish-red area and grayish-yellow halo around it. Second stage of the disease.

unsatisfactory because patient could not be induced to fix steadily.)

CASE 2.—Mrs. Ben B., aged 36. Never had any complaints about her eyes.

Vision R. $\frac{5}{10}$; L. $\frac{5}{10}$.

Retinoscopy under homatropin: R. + 3.5s ⊂ + 0.5 axis 15°; L. + 3.5s.; with this correction: R. $\frac{5}{6}$?; L. $\frac{5}{6}$.

*Ophthalmoscopic Examination:* R. E. Disk well defined, round, not elevated; very small physiological, central, excavation. Slight increase of diffuse, gray pigmentation at temporal margin of disk. In macular region dark cherry-red round area, ⅓ disk diameter wide, showing no details, apparently occupying area of fovea; only here and there brilliant reflexes will show up over area and at margin of it as patient moves the eye. On the temporal side of the macular area, about 1½ disk diameters to temporal side of above mentioned spot, a group of yellowish-white spots of different size and different brightness with indistinct edges, lying distinctly behind retinal vessels: colloid bodies. Balance of fundus normal.

L. E. Disk well defined; small, round, shallow, central excavation. A trace of irregular gray pigmentation at temporal margin of disk. In macular area a spot as in right eye with almost exactly the same characteristics, and similarly also there is also a group of yellowish spots at temporal side of the macula.

In each eye the oval reflex at the edge of the macula will show up occasionally with the Gullstrand but never complete in any position; no difference in level between central red area and surrounding retina could be detected with binocular Gullstrand.

Field examination of each eye as in Case 1.

Wassermann and Luetin tests could not be made.

CASE 3.—Herbert P., aged 26, first called January, 1912. No difficulty when at school, learned to read and write Hebrew; was known for his good distance vision and was often called on to recognize who was coming at a distance. In 1910 entered drygoods store as clerk, being rapidly advanced; 1911 noticed some difficulty matching colors, having been in the meantime advanced to the dressgoods department, began noticing that he could not recognize his street car or any one coming; sight better at times, very dim at other times; was not aware did not know colors. No lues.

V. O. D. $\frac{5}{9}$?; V. O. S. $\frac{5}{9}$?.

Retinoscopy under homatropin: R. + 0.5s ⊂ + 0.5 axis 90°; L. the same; with this correction: R. $\frac{5}{9}$ +; L. $\frac{5}{9}$ +.

*Ophthalmoscopic Examination:* R. E. Disk almost round, well defined, not elevated; slight absorption and irregularity of pigment at temporal margin of disk. In macula large oval or pear-shaped, horizontal, slightly irregular area, beginning about one and a quarter to one and a half disk diameters from temporal margin of disk, extending in a horizontal direction about one and a half disk diameters; in a vertical direction less than one disk diameter wide; area to be divided into two distinct parts: an outer large halo-like, and a central comma-like portion.

(*a*) Outer, peripheral portion, lighter than surrounding fundus; grayish-yellow, marbled; very lively reflexes in front make exact examination of the area itself impossible, but one can make out that its peculiar color is due to the apparent absorption of pigment in minute spots between which are granules of pigment; two or three large spots of pigment and one fairly large colloid body can also be seen in this halo. (*b*) Central portion, comma-like, or comparable to the head of a tænia; fairly well defined, brownish- or purplish-red; thinner and gradually blending into peripheral area on its temporal aspect.

Balance of fundus slightly tigered; media clear.

L. E. Similar picture as in right eye only the central area is more dark-red, more saturated, the edges more sharply defined, and in the head of the "tænia" there is a constriction between the head and neck of the tænia-like area. Patient could not be induced to sit for painting of left eye.

Field examination with round disk of 5*mm* diameter showed almost normal limits for white and detected his inability to distinguish colors. Examination with Holmgren and Oliver wools proved his unmistakable color blindness.

February, 1912, Wassermann was positive. Was given Salvarsan injection and later mixed treatment, which he has continued with interruptions until date.

There is hardly any doubt that these changes in the macular region are not due to acquired disease, but must be looked upon as the result of some congenital weakness of the macular region. The striking symmetrical arrangement, or as Stargardt calls it, "the photographic trueness of the macular changes," at once suggest the idea of a congenital and hereditary factor. And while it is well possible, and has even been reported, that choroidal or retinal disease will occasionally produce symmetrical changes in both eyes of a patient, the fact that these changes are found in three members of a family

and each time in a symmetrical arrangement, makes it almost impossible that the condition could be due to anything but to a congenital or inherited weakness of the region. This view is further strengthened by the fact that other marks of ocular inherited stigmata are found among the members of the family. As such we may count the color-blindness of all the brothers examined and the posterior polar cataract in the mother of the patients, Mrs. P.; this form of opacity of the lens has been reported in several members of a family and is considered as significant of other affections of the eyes such as pigment degeneration, choroiditis, etc.

The chief interest in these cases centers around the foveal area with its peculiar, dark-red color and the different shape it assumes in the three patients, though in each instance it appears exactly symmetrical in the two foveæ of the individual patient. When occurring alone, as in the case of Mrs. B., it must be considered as the first stage or slightest degree of macular degeneration. As the disease progresses, a comparatively mild atrophy of retina and choroid develops immediately around this foveal area, as in Case 3, Mr. P. In a still later stage, when the disease has further advanced, foci of atrophy show up in other parts of the retina, as in Case 1, Mrs. D., and it is interesting that those peripheral foci, though apparently younger, have led to more extensive absorption of choroid and retina than the older, more central ones.

Several points worthy of interest stand out in these cases.

Oatman emphasizes the point that no case of this disease had been found among Jews. Here we have for the first time the disease in three members of undoubted pure Jewish extraction. It is possible that the O. family in Darier's paper is also Jewish, because it is mentioned in Charles O.'s history that he had been taught Hebrew by his father.

The onset of the trouble in familial macular degeneration is generally placed at the age of puberty, if the macular disease is the only affection of the patient, and at the age of about seven when the macular condition is accompanied by cerebral degeneration (affection of the mentality). About the patients here reported, we have no exact data by which the onset of the trouble can be marked: Mrs. B. has no complaint about her eyes at all, and Mrs. D., in spite of her very advanced fundu

condition, has pretty fair vision. Only in Case 3, Mr. P., have we a right to assume that the change in the macula began about the time when he first noticed his distance vision failing, etc., when about twenty-two years old, so that in this instance the symptoms appeared later than in the other cases reported.

Macular degeneration generally leads to very severe disturbance of central vision, whether of the purely macular or of the maculo-cerebral type. The patients above reported all show exceptionally good vision in spite of the high error of refraction, as in the case of Mrs. B., and in spite of the rather advanced condition of the fundus, as in the case of Mrs. D.

Macular degeneration is to be considered a progressive disease, and our patients illustrate the different stages in the progress of the trouble, even showing a fresh focus as in the case of Mrs. D. In all the cases reported the progress is a comparatively rapid one. Judging from the present vision of our patients, even of the most advanced case of Mrs. D. whose vision has not materially changed since she first came under observation eight years ago, the disease in this instance seems to be rather slowly progressive, more benign. Possibly this is due among other things to the fact that two of the patients are women and consequently, as Stargardt points out, less liable to rapid advancement of the disease because of more hygienic mode of living, such as abstention from tobacco and alcohol. On the basis of this theory it is possible that the heavy smoking of Mr. P. may have made his subjective symptoms manifest at the age of twenty-two years. During a recent examination in February, 1916, Mr. P.'s fundus was found unchanged. For certain reasons his vision could not be taken, but he emphasized the fact that his vision was possibly better than formerly. This may be due to the fact that he has been taking mixed treatment and has abstained from tobacco and alcohol, as per instructions.

Stargardt emphasizes that in cases of purely macular degeneration consanguinity of parents had not been noted. Our cases distinctly represent the purely macular type; still their parents are children of two brothers.

It is interesting that no scotoma could be detected in any of the patients. While this may be due in part to the patient's

lack of attention, similar instances may be found in the literature. Deutschmann, for instance, reports his case of a very intelligent young man with bilateral congenital hole in the macula but with no central scotoma, and alludes to similar instances in the literature.

Any attempt to interpret the objective findings would be based entirely on theoretical grounds. Still the color of the central foveal area challenges attention. A similar color of this fundus region is found in amaurotic family idiocy and in obstruction of the central retinal artery. This color is said to be contrast color due to the pallor of the surrounding retina and due to the fact that the retina is thinner at the spot, allowing the choroid to shine through; a similar color may also be seen in the typical hole at the macula of Haab. In our patient, Mrs. B., there is absolutely no question of contrast with the surrounding retina, because it appears perfectly normal in this neighborhood and only possibly more pigmented. In the other two instances, Cases 1 and 3, the details of the coloring, such as saturation and form, distinctly exclude the question of contrast though the surrounding retina is grayish-yellow. Special attention directed to this point failed to discover, even with the binocular Gullstrand, any difference in level, even in the simplest case of Mrs. B. that difference in level which is easily seen even with the ordinary ophthalmoscope in typical cases of Haab's macular affection.

Still the red color of this region may be explained as due to slight thinning of the retina which took place less abruptly, less well-defined, but in a more gradual and sloping manner. In consequence of such thinning no crater-like, punched-out hole with marked difference of level was produced, but the whole area was converted into a more flat, saucer-like depression with sloping sides. The absence of the normal foveal reflex in our cases would possibly prove that the normal foveal pit giving rise to the normal reflex has been converted into a flat, shallow depression with sloping sides, as assumed above.

The peculiar, grayish-yellow color of the area around the central reddish spot is undoubtedly due in part to the minute foci of absorption of pigment cells and to some of the pigment migrating into the innermost layers of the retina; possibly there also exists a slight cloudiness of the retina in this region.

A detailed examination of this area was made difficult by the lively reflexes of the retina in front of it.

The peculiar shape of the macular foci can best be explained at the present when we remember that the anatomical condition of the choroid and retina of the macula is different from that of the area surrounding it. If we assume that slight vascular changes in this area are brought about by some congenital factor, the picture, as seen in these cases, can easily be explained. The cases reported by Pusey would strengthen such assumption.

As to the causative factor of the disease our cases offer no suggestion. There is no reason to assume that syphilis of the parents played any rôle, but the evidence against consanguinity as having some share in the etiology is accumulating. Because the atrophy of these structures is quite advanced when the patients call for help and because of our present ignorance as to the etiological factor, no hope from any treatment can be extended to these patients.

It remains for further observation to establish the exact pathology of this disease, to detect its etiology, and to outline the different types that make up the picture of macular degeneration.

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# THE MEDICAL SIDE OF GLAUCOMA.[1]

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*(With two diagrams in the text.)*

IT must be admitted that there is an underlying general cause for glaucoma, yet the connection between glaucoma and general disease is so vague that attention by ophthalmologists has practically been centered only upon the local changes in the eye. This neglect has, in recent years, changed, and the general condition of glaucoma patients has been the subject of considerable investigation. The question is somewhat simplified if we realize at the very beginning that under the term "primary glaucoma" a number of conditions are collected, extremely dissimilar in many features, particularly in etiology, though increased intraocular tension is a common symptom to all. We can furthermore exclude from this consideration cases of glaucoma due to disease of the retinal vessels, the so-called hemorrhagic glaucoma (thrombosis of the central retinal vein).

Next it is desirable to briefly explain that normal intraocular pressure is produced by the blood pressure, which, in turn, is the source of the fluid within the eye. This fluid results not so much from a secretory process but from a transudation of a peculiar kind in which, under normal conditions, certain constituents of the blood serum are retained, as a difference in the amount of albumen contained in blood serum and in the aqueous humor will show. The production of the ocular fluids is to a great extent dependent upon blood pressure, as

[1] Read by invitation before Ohio State Medical Meeting, Cleveland, May 18, 1916.

is the ocular pressure. Wessely has examined experimentally the dependence of the intraocular pressure on the blood pressure. He found that the pulsation of the ocular pressure presented a distinct curve a moment later than that of the carotid. If the blood pressure is increased, the ocular pressure is also increased, though to a lesser degree on account of certain regulations, such as the reduction of blood pressure in smaller vessels, increased lumen of the vessels, elasticity of the eyeball capsule, and degree of distension of the eyeball. Contrariwise, the reduction of blood pressure causes a depression of the ocular curve. From this dependence of ocular pressure on general arterial pressure it seems natural to infer that glaucoma could be readily explained by pathologically raised blood pressure, and that there must be a close connection between glaucoma and disease of the cardio-vascular and renal systems in which high blood-pressure is so important a symptom.

*Arterial Tension in Glaucoma Patients:* I examined fifty cases of primary glaucoma of which I had kept careful records, and found the blood pressure to average about 150; it was 140 in patients under sixty years old and 150 in those over sixty.

Kümmel (*Graefe's Arch.* vol. lxxix., No. 2) examined seventy cases of glaucoma and found the blood pressure in those below sixty was 163.2; over sixty, 169.3; together 165.7. The normal pressure as found in ninety cases without glaucoma and of about the same age showed an average of 145.2; in persons over sixty the average was 152.85, and in those under sixty it was 138.6. This shows that in $\frac{5}{6}$ of the cases there was an increase of blood pressure.

This author also found a difference in arterial pressure according to the variety of glaucoma. Fourteen cases of acute glaucoma showed an average pressure of 177. This is not unexpected, as a disturbance in the vascular system is present. The pressure varied between 160 and 245. The conditions were similar in the subacute cases; in eighteen the average was 175.2. It has been pointed out particularly by the French authors, that blood pressure is increased in the inflammatory forms. The arterial pressure in chronic glaucoma was found increased only to a slight degree (160.6) and lower than in the inflammatory forms, though these cases are usually in old people. In thirty cases the average pressure

was 153.3; in seven of these the pressure was below the average 145*mm*, and four presented other symptoms of diseases of the cardio-vascular system or of the kidneys.

Krämer (*Graefe's Arch.*, vol. lxxiii., p. 349), on the other hand, found no direct connection between blood pressure and glaucoma, and in his investigations there was no difference in blood pressure between the acute and chronic forms of glaucoma. Sattler (*Berl. klin. Wochenschr.*, 1913, Nos. 49 and 50) also could find no regular increase in the blood pressure of his glaucoma patients. He adds that persons suffering from high blood-pressure are not specially prone to glaucoma and usually show other eye lesions, particularly often associated with local disease of the vessel walls. He has seen no glaucomatous attacks attributable to increased blood-pressure from muscular exercise. On the other hand, the exhaustion and heart failure which follow over-exertion, mental depression and weakening disease such as influenza, are frequently associated with glaucomatous attacks. Löhlein (*Zentralblatt f. ges. Ophth.*, 1914) does not believe that disturbances of the cardio-vascular system can be regarded as an essential factor in glaucoma. In many pronounced glaucomas we do not find high blood-pressure or venous congestion of head and eye, and the absence of glaucoma in conditions with high blood pressure (nephritis) and blood congestion to the head show that there must be local predisposing causes.

*Ocular Tension in General Arteriosclerosis:* The examination of the ocular tension in general arteriosclerosis has given valuable information on the question of this relation. Thus Moore (*R. L. O. H. R.*, xx., 1915) examined thirty-eight patients, the subjects of general arteriosclerosis, in whom the eyes showed no abnormality other than sclerosis of the retinal arteries; and found that such variations in the ocular tension as occur are not caused by nor associated with a like variation in general blood-pressure. He compared the intraocular tension of patients with a general blood-pressure of 220 or more and those with a blood pressure of 160 and below, and found an insignificant difference. Krämer (*l. c.*) also examined fourteen patients with chronic vascular and renal lesions who presented a very high blood-pressure but in whom the ocular tension was normal.

Priestley Smith (*Ophthalmic Review*, 1911), with characteristic acumen, has drawn attention to the important fact that chronic excess of pressure in the radial or carotid arteries is no evidence of excess of pressure in the capillaries that secrete the intraocular fluid; on the contrary, it is usually associated with increased resistance in the arterioles and insufficient capillary circulation.

Starling and Henderson found that a considerable rise in the general blood-pressure did not with any constancy produce a permanent rise in the intraocular tension, and Moore concludes that in some cases of general arteriosclerosis with a high general blood-pressure, the pressure of the ocular vessels is below the normal. Intraocular tension is presumably dependent on the condition of the ciliary arteries for which the ophthalmoscopically visible retinal vessels are no guide, as a considerable independence is manifested in the degree in which the various systems of vessels in the eye are involved.

*Ophthalmoscopic Changes:* It has seemed to me worth while to examine the retinal vessels for sclerotic changes in the above-mentioned series of fifty patients to determine the value of ophthalmoscopic findings in glaucoma. In the forty cases of chronic glaucoma arteriosclerotic changes in the retinal vessels were present in eight, and these were all mild in character. In the ten cases of acute and subacute forms (the congestive type), arteriosclerotic changes were present in eight; in the other two a satisfactory ophthalmoscopic examination could not be made. I was particularly struck by the frequent and very marked changes found in the acute glaucomas after the attacks were past; some of the arteries were nearly obliterated and sheathed with broad white lines, and the disks were pale.

*Vascular Changes in the Eye:* The question of local vascular changes in the eye is an important one. General vascular disease surely indicates changes in the more delicate vessels in the eye. The results of pathological examination of ocular vessel changes in glaucoma are varying. Bartels (*Zeitschrift f. Augenhlk.*, vol. xiv.) acknowledges the frequency of these changes after a critical examination, though he does not think that these changes are sufficient to be specific and are not greater than the sclerotic changes which are to be expected.

Arteriosclerosis diminishes in a distal direction; thus the changes must be more pronounced in the orbit than they are in the eye. In hemorrhagic glaucoma Kümmel demonstrated marked changes in the ciliary arteries back of the eye.

*Nephritis.* Kümmel found in 45% of his cases changes dependent upon nephritis, albuminuria with relatively few casts. Joseph, by means of the methylene blue method, showed a relative kidney insufficiency. Sulzer and Ayrignac (*Annales d'oculistique*, vol. lc., p. 245) have recently studied metabolic disturbances in glaucoma. They found a reduction of liver function and relative impermeability of the kidneys producing an insufficient using up of albumen. This results in a form of arthritism which is related to increased pressure from increased secretion in an eye predisposed to glaucoma.

*Nasal Empyema:* The close nervous relation of the nasal accessory cavities and the eye has brought glaucoma in etiological relationship to nasal empyema in the opinion of certain observers (de Lapersonne, Fish, etc.). There is in fact a close association between the second division of the trigeminal nerve and the wall of the sphenoid sinus. Sluder has drawn attention to the rôle of spheno-palatine ganglion in neuralgia and has relieved the pain of glaucoma by anæsthetizing this ganglion(A. E. Ewing, *A. J. O.*,Dec., 1908). Ludde reports on the reduction of intraocular tension after cocainizing this region of the nose. I have tried to observe some change in ocular tension after anæsthetizing the region of this ganglion in glaucoma cases, but without any success. In the cases where nasal empyema was associated with glaucoma no change whatever was observed in their ocular tension after the nose had been operated upon.

*Nervous Disturbances:* There is no question but that vasomotor irritability, migraine, affections of the V. nerve, etc., are symptoms which frequently affect glaucoma patients; yet the exact nature or cause of these possible factors in the production of glaucoma is not definitely known. The vasomotor fibers are a part of the sympathetic nervous system which in turn is closely related to the ductless glands. Experiments have proven (Priestly Smith, *Oph. Rev.*, p. 5, 1911) that the intraocular pressure may be raised considerably by irritation of the sympathetic and still more by irritation of the V. nerve,

the result in each case being probably due to the action of vasodilator fibers. Parsons and Snowball (*R. L. O. H. R.*, xv., 1903) have shown experimentally that stimulation of the sympathetic produces a reduction in tension owing to a constriction of the ciliary vessels. Unfortunately much of the experimental work on these nerve reactions is contradictory. Dubois (*K. M. A.*, 1912, p. 601) observed a case of acute glaucoma in the course of a typical herpes zoster of the first branch of the V. nerve. He believes that both were caused by trophic and vasomotor nerve lesions. Bradburne (*Ophth. Rec.*, p. 169, 1909) saw a case of herpes zoster ushered in by an attack of glaucoma. Schmidt-Rimpler (*Recueil d'opht.* xxxii., p. 164) found after extirpation of the Gasserian ganglion that the ocular tension reduced to 10*mm* with narrowed palpebral fissure. In the blood serum of glaucomatous patients Kleczkowski (*Kl. M. A.*, Oct., 1911, p. 417) discovered adrenalin. This observation, however, was not confirmed by other investigators. A striking symptom in glaucomatous eyes is the atrophy of the iris which is often circumscribed and superficial. Bistis (These Archives, 1915) has published a case of heterochromia with signs of paralysis of the cervical sympathetic, and was able to show in rabbits that extirpation of the cervical sympathetic was often followed by this change. Cannot this symptom therefore be brought in relationship to a sympathetic disturbance? Adrenalin is said to dilate the pupil in an eye with sympathetic paralysis. After the subconjunctival injection of novocain and adrenalin in glaucoma operations the resulting dilatation of the pupil has surely been generally noted. These fragmentary reports, I think, suggest an involvement of the sympathetic nervous system in glaucoma; it is, of course, impossible to say whether this lesion is the primary one or not.

The following case possibly illustrates this involvement.

M. H., age 27, on Nov. 3, 1915, came to the Eye Hospital stating that five weeks ago he noted that the sight in the right eye was very poor. This was followed by some obscure pain, and the sight in both eyes became progressively worse. On examination V. = R.: H. M.; L. $\frac{20}{60}$. To. R. 49; L. 30. Blood pressure 105. Urine neg. Wassermann neg. Fields very much contracted (see Fig. 1). The ocular tension and

blood-pressure were taken twice daily, but the variations did not seem to be related (see Fig. 2). Optic nerves show

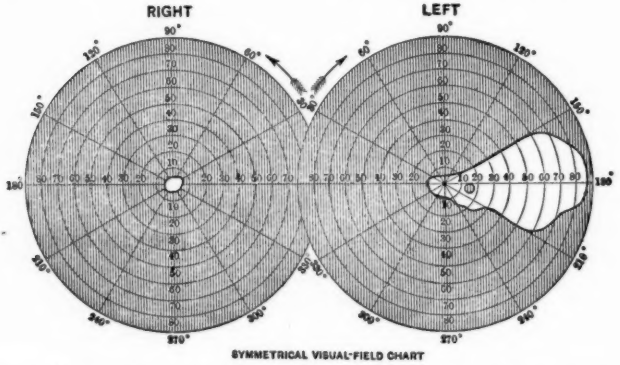



FIG. 1—Visual Fields of M. H. Chronic Glaucoma, October 24, 1915. O. D.: V= $\frac{1}{200}$ T 33. O. S.: V=$\frac{20}{50}$ T. 20.

a deep cup and atrophy. He was admitted to the hospital and subjected to a most careful general examination. This

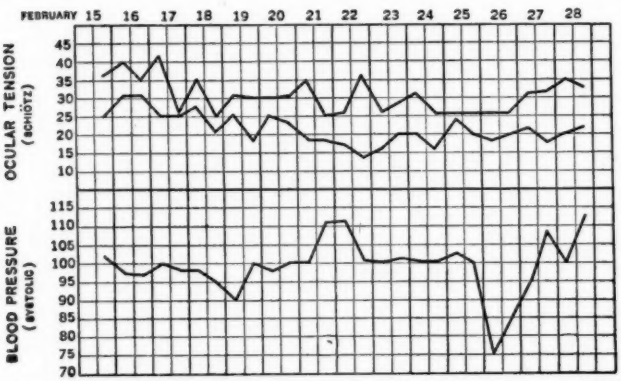



FIG. 2.—Comparison of blood pressure and ocular tension readings taken twice daily.

proved negative; except for hyperthyroidism, the thyroid gland was enlarged, uniform, and soft. Vasomotor irritability. Tachycardia. He was put on various glandular

extracts without any distinct change, the ocular condition has remained stationary.

This case reminded me particularly of an article by Lagrange (*Arch. d'opht.*, vol. xxxiii.) on the "Prognosis of Chronic Glaucoma" in which he says that the neuropathic glaucomatous individual presents deep trophic disturbances, the atrophy and degeneration of the optic nerve are most marked, and in his opinion one must distrust the optic disks whose pallor is out of proportion to the depth of the excavation.

In conclusion, if I should be asked to classify the forms of primary glaucoma which I have attempted to describe, I should divide them into two groups: circulatory and nervous. The circulatory group would be characterized by congestive attacks, by retinal vascular changes, by local changes in the eye which favor increased tension, such as small eyeball, age of patient, etc., and the nervous variety which shows dysglandular disturbances, no arteriosclerosis, and affecting any type of eye, myopic as well as hypermetropic. The treatment in the first group is operative, and of greatest importance is the attention to the general health; in the second group, operation is indicated in some; our efforts at general treatment must await further knowledge of dysglandular affections.

# TUBERCULOSIS OF THE RETINAL VESSELS.

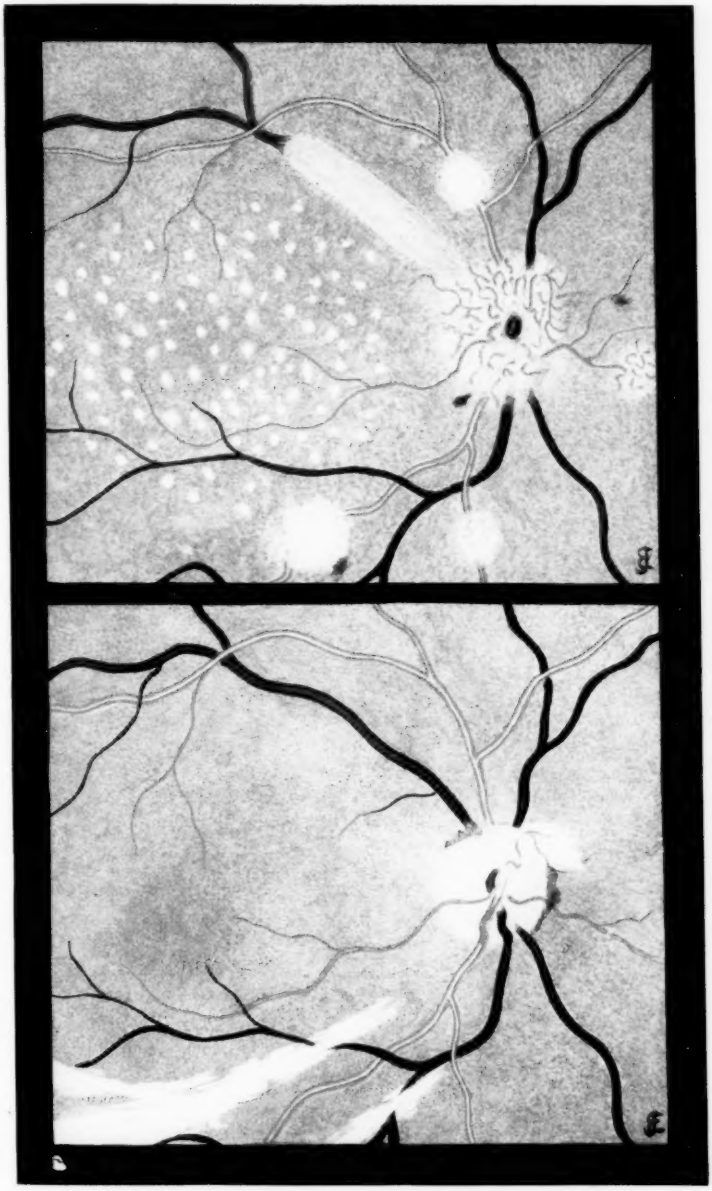
By EDWARD JACKSON, M.D., Denver, Colo.

(*With illustration on Text-Plate XXVIII.*)

FOR the practical management of cases the diagnosis of intraocular tuberculosis must rest on the clinical picture revealed by the ophthalmoscope, and the reactions to special tests. Studies in pathologic anatomy can give a definite basis and significance to the ophthalmoscopic findings. But they are not available when the case presents for diagnosis and treatment. Practical advances in regard to these conditions must depend on a definite and complete appreciation of the ophthalmoscopic symptoms to which they give rise.

Tuberculosis starts as a local disease; and as regards anatomic lesions, unlike syphilis, it probably remains a local disease in many cases. Some of the general symptoms commonly associated with it arise from mixed infection and the general effects of pulmonary involvement. The earlier known and most common manifestation of the disease is that systemic resistance manifest in the tuberculin reactions. Even this is not constant, or it is concealed by unknown conditions, as we have learned by failures of the various tuberculin tests. Probably with primary intraocular tuberculosis, the conditions surrounding the local lesion especially oppose the development of any general manifestations whatever. Von Hippel reported a case of tuberculosis in which a febrile reaction was obtained only from the sixth injection of old tuberculin in dose of 5*mg*. I have seen a case of intraocular tuberculosis in which no reaction was obtained from a first diagnostic injection of tuberculin; and a case of general bilateral corneo-scleral-uveal tuberculosis, that later developed

ILLUSTRATING DR. JACKSON'S ARTICLE ON "TUBERCULOSIS OF THE RETINAL VESSELS."



**Tuberculosis of Retinal Vessels.**
**Upper—Early stage. Lower—Late stage.**

tuberculous meningitis, in which the von Pirquet test proved completely negative. In the case on which this paper is largely based the von Pirquet test proved negative, although an injection of tuberculin gave systemic and focal reactions. This does not show that the tuberculin diagnostic tests are not of great value. But it does emphasize the importance of getting as correct and definite a conception as possible of the ophthalmoscopic symptoms, an importance that justifies the presentation of the accompanying sketches of them.

Within the last two years the writer has seen five cases presenting the ophthalmoscopic appearances of retinal tuberculosis. Three of these, in which the diagnosis was fully supported by the results of tuberculin injection, were under regular observation for many months. Two of these have been reported last year (*Trans. First Colorado Ophth. Congress*). The case on which the accompanying plate is based has been under observation for over eighteen months; and throughout nearly the whole period presented conditions exceptionally favorable for ophthalmoscopic study. Vision in the one eye affected was at one time reduced to counting fingers at six inches, but has risen again to 0.8, and the eye has remained without fresh lesions about a year. The title tuberculosis of the retinal vessels has been used because in these cases the symptoms and visible lesions are strikingly connected with the blood vessels.

In all of these cases there was localized clouding of the vitreous not definitely connected with any retinal lesion. In the case here represented there were these vitreous clouds with indefinite edges and dense centers that were not connected with any hemorrhage or visible vascular lesion. These were well inside the vitreous and seemed quite surrounded by relatively clear vitreous. In every case there was also from time to time diffuse clouding of the vitreous from hemorrhage. But in the case here represented the general clouding occurred but once near the beginning of the disease.

In every case there were hemorrhages. In the one here illustrated these hemorrhages, except the first, were small and confined to the retina. It was this feature that made the case so favorable for ophthalmoscopic examination. In all the other cases the hemorrhages repeatedly broke into the vitreous,

giving the typical picture of recurring retinal hemorrhage in young persons.

A most striking ophthalmoscopic appearance in the case here represented is that of the rounded spots smaller than the optic disk with indefinite margins; at first light gray in color, as the one above the disk, later becoming vascular, as the one to the nasal side of the disk; always located on one of the larger vessels and finally disappearing to leave the region involved almost or quite normal. In the beginning the patch had some resemblance to a choroidal tubercle, but was whiter, less yellow in color. The mass completely concealed the large vessel that passed through it. In no case did any alteration of pigmentation follow. The impression gained was that each of these spots represented an exudate involving the vessel wall and the adjoining vitreous. A lesion that might be provoked by the presence of a colony of tubercle bacilli, but which probably would not exhibit the structure of a fully formed miliary tubercle.

The most obvious alteration in any of the vessels was exhibited by the upper temporal vein. Toward the periphery this appeared normal in color and contour, or slightly dilated. But at a certain point it abruptly widened out to full four times its normal diameter, and became gray tinged with the color of the normal venous blood column. Its sides were nearly parallel and comparatively straight. It appeared to be a swelling of the vessel wall with dilatation of the vessel lumen. This condition changed slowly during five or six months; and then returned to the normal. This lesion had scarcely begun to undergo involution when the rounded spots above referred to, and the white dots next to be mentioned, had almost entirely disappeared.

An interesting part of the ophthalmoscopic picture is the small white spots in the macula. These were generally less brilliant than the spots of renal retinitis or papilledema, although they approached that appearance. The largest were two or three times the diameter of the largest normal retinal vessels; their edges faded gradually into the normal fundus surrounding them. They developed gradually and faded away gradually, some more rapidly than others, but all were evolving or undergoing retrogression together. They appeared

to be the manifestations of some general condition affecting the nutrition of all that part of the retina; and not separate centers of pathologic activity arising independently of each other, like local infections.

Spots of this kind were noticed in two of the five cases, and they have been noted by other observers, so that they must be regarded as an important ophthalmoscopic symptom of this disease. Knapp (*Trans. American Ophth. Soc.*, vol. xiii., p. 486) recorded them in one of his cases, although the radiating figure shown in his plate representing the case bears more resemblance to the star of renal retinitis than did the grouping of these spots seen in my patients. These spots had disappeared entirely about the time that central vision had risen to 0.7. They left no trace of altered pigmentation.

About the time the diagnostic injection of tuberculin was given, in the case represented and in one of the other cases while under observation, the optic disk became hidden in a gray non-vascular haze, with indefinite edge, that a little later became vascular. The appearance came to resemble that of an increasing papilledema, except that the central retinal vessels were not choked, and the boundaries of the swelling were less abrupt. This swelling showed no tendency to give place to post-neuritic atrophy, but gave rise to a permanent condition of white connective tissue but poorly supplied with vessels, but sometimes showing on its surface large new-formed vessels typical of retinitis proliferans. In the case shown this scar tissue reached forward 5 D. in front of the disk. The strands extending toward the lower periphery of the fundus were less prominent. In other cases the scar tissue was much more extensive, stretching forward to the extreme periphery of the fundus, and from 10 to 20 D. in advance of the normal surface of the retina.

The lesions showed marked focal reactions to tuberculin injections. In the cases shown the swelling over the nerve head became highly vascular, changed color completely within forty-eight hours after the diagnostic injection that raised the body temperature one degree, and minute hemorrhages occurred after other injections. In another case, on two occasions within forty-eight hours after therapeutic injections, large vitreous hemorrhages occurred, greatly reducing the

vision that had been regained, and which has since risen to 0.4 and remained there for three months.

Another patient without active lesions for over nine months had vision slowly improving up to 0.4. She had become pregnant, went to term, bore a healthy child, and nursed it. But when the child was three months old the trouble recurred and a hemorrhage reduced vision in one eye to moving objects. None of these patients has shown marked evidence of pulmonary tuberculosis, although I have seen a case in which the ocular lesions appeared late in the disease; but all showed moderate anæmia, and some departure from robust health.

The clinical picture that it seems important to fix in relation to retinal tuberculosis includes these features: Vitreous opacities, recurring retinal and vitreous hemorrhages, enlargement of the retinal veins, the local lesions described as associated with large retinal vessels, white spots in the macula in some cases, optic neuritis, and retinitis proliferans as a terminal condition; the earlier lesions giving reactions to tuberculin injections, and their involution favored by tuberculin therapy.

# OCULAR ANAPHYLAXIS: I. THE REACTION TO PERFUSION WITH SPECIFIC ANTIGEN.

By ALAN C. WOODS, M.D., PHILADELPHIA.

(From the John Herr Musser Department of Research Medicine, University of Pennsylvania.)

*(With three line illustrations in text.)*

THE observations of Uhlenhuth (1), as early as 1903, on the singularity of lens protein as compared to other body protein, led to the establishment of the facts (*a*) that lens protein could act as a foreign protein to the homologous animal, and (*b*) that it lacked species specificity. During the experiments which resulted in these conclusions certain local ocular phenomena were observed, which turned the attention of many investigators and ophthalmologists to possible anaphylactic phenomena which could be elicited in the eye itself. As a result of these researches it became clear that local anaphylactic phenomena could be produced in the eye by the intraocular injection and reinjection of foreign protein—an observation in line with that of Arthus (2) in 1903, that cutaneous lesions resulted at the site of injection, in rabbits repeatedly injected with horse serum.

Nicolle and Abt (3), in 1908, immunized guinea pigs by repeated intraperitoneal injections of inactivated horse serum. By anterior chamber injection of the same antigen thirty-two days after the first intraperitoneal injection, they obtained marked ocular inflammatory symptoms, iritis, hypopyon, and conjunctivitis. Sattler (4), in 1909, showed that the injection of heterologous serum in the eye could in itself give inflammatory symptoms, which appeared about five days after injection and persisted a varying length

of time, gradually subsiding. This observation somewhat obscured the results obtained previously by the intraocular injection of foreign protein in sensitized animals, and in subsequent work the time of appearance of symptoms was used as a guide to differentiate true anaphylactic symptoms from those dependent upon the injection of foreign protein.

Krusius (5), in 1910, sensitized guinea pigs by intraocular injection of cow's serum, and later obtained typical anaphylactic death by intracardial injection of the same antigen. Intraocular injection of cows' serum in animals previously sensitized by subcutaneous injection of the same antigen also gave prompt and marked ocular inflammation. The same phenomena were observed by using homologous lens protein, and slight, but definite, reactions were obtained by sensitizing animals by discission of the lens and subsequent intracardial injection of homologous lens protein. Likewise, discission in animals previously sensitized by subcutaneous injection of homologous lens protein gave slight ocular inflammation. Romer and Gebb (6) repeated this work in 1912, using as an indicator to anaphylactic reactions the drop in temperature following reinjection. They confirmed Krusius's work with cows' serum as antigen, but failed to substantiate his results obtained by discission.

Kummel (7), in 1910, by intraocular sensitization, and either intraocular or intravenous reinjection, and by subcutaneous sensitization and intraocular reinjection, obtained ocular inflammation. He concluded (*a*) that animals could be generally sensitized by intraocular injection, and (*b*) that the eye could be sensitized by subcutaneous injection. Further, sensitization of one eye and reinjection in the second eye gave inflammatory reactions in the second eye, which were confined practically to the uveal tract.

In 1911, Wessely (8) reported his well-known experiments upon keratitis anaphylactica. Rabbits were sensitized by intralamellar corneal injection of heterologous serum. The traumatic injury incident to this injection quickly subsided, and the cornea remained entirely clear for fourteen days, when corneal opacities with pericorneal injection developed. This reaction Wessely supposed to be anaphylactic in origin and due to the slow absorption of antigen. This reaction in turn subsided and after fourteen days the cornea of the second eye was similarly injected. A violent parenchymatous keratitis at once developed, which was termed "keratitis anaphylactica."

In 1913, Szily (9), with the collaboration of Arisawa, repeated this entire work. They confirmed the delayed ocular inflammation following the single injection of foreign serum observed by Sattler in 1909. Prompt ocular in-

flammation was obtained by reinjection of foreign serum in eyes previously sensitized by similar injection. Intravenous injection of specific antigen in animals in whom one eye had been previously sensitized by intraocular injection gave the usual inflammatory phenomena, but in addition they observed a prompt contraction of the pupil, in the sensitized eye alone, immediately following the intravenous reinjection. Intraocular injection in animals previously sensitized by either intravenous or intraperitoneal injection gave marked inflammatory symptoms. Similar phenomena were observed using as antigen foreign lens protein and uveal emulsion. Szily and Arisawa confirmed the results obtained by Wessely by intracorneal sensitization and reinjection, and further showed that intravenous reinjection in animals, previously sensitized by intracorneal injection, gave a keratitis, with the prompt contraction of the pupil before observed. In all their experiments, the phenomena were dependent upon the reinjection of the specific antigen—injection of cows' serum giving no results in animals sensitized with horse serum.

From the experiments cited it is evident that ocular inflammatory reactions (an iridocyclitis on intraocular injection and a parenchymatous keratitis on intracorneal injection) can be produced under the following conditions: I. By the reinjection with specific antigen of eyes previously sensitized by intraocular injection. II. By intravenous injection of specific antigen in animals previously sensitized by intraocular injection. III. By ocular injections in animals previously sensitized by either subcutaneous, intraperitoneal, or intravenous injection.

However, these observations are open to several criticisms. First, foreign protein has in itself a deleterious action upon the tissues of the eye, and secondly, a certain amount of trauma necessarily accompanies intraocular injection. The tissues of the eye, and especially the ciliary body, are very delicately organized and highly susceptible to injury, and injury is liable to be so far-reaching and extensive that the interpretations of inflammatory lesions, following intraocular injection, are somewhat difficult.

Furthermore, even though these phenomena are probably truly anaphylactic in nature, possible anaphylactic phenomena observed in the human eye must be dependent

upon a different mechanism. Ocular sensitization must be part of a general sensitization, and antigen to evoke the anaphylactic reaction must be carried to the eye either by the blood or lymph. In other words, sensitization and intoxication in man scarcely ever occur by actual injury or injection of the eye. The phenomena of sensitization and intoxication must be through a vascular system. The phenomena observed by experimental ocular injection and intravenous reinjection, and by general sensitization and intraocular reinjection each partly meet this objection, but no ocular anaphylactic phenomena have so far been observed in animals generally sensitized and reinjected intravenously. The reasons for this are, apparently, that the stormy general symptoms in animals so sensitized and reinjected mask any ocular response, and the prompt death of the animal in anaphylactic shock makes protracted observation of the eyes impossible.

With the idea of ascertaining if anaphylactic phenomena could be elicited by antigen carried to the eye via the blood stream, in animals previously sensitized by intraperitoneal injection, and of making a somewhat protracted study of such symptoms, the work here reported was undertaken.

## TECHNIQUE.

Dogs were sensitized by intraperitoneal injection, and after an interval of from fifteen to twenty-five days the eyes were perfused with defibrinated normal dogs' blood and Ringer's solution, in the proportions of four to one, respectively. To this perfusion fluid specific antigen was added after preliminary observations had been made, and the perfusion continued, as a rule, for a period of three hours. In order to successfully perfuse the eyes, it was found necessary to perfuse the entire head. Preliminary experiments, in which the head was perfused with normal salt solution containing a suspension of ultramarine blue, showed that the distribution of the dye to the eyes was complete and thorough.

With the apparatus used, if the operative technique was perfect, the winking reflex continued throughout the first hour. In several perfusions a slight reaction of the pupil to light was obtainable during the first few minutes of the perfusion.

*Apparatus.* The perfusion apparatus used is shown in the accompanying diagram (Fig. I). Pressure was obtained from a Ludwig constant pressure apparatus. The perfusion fluid was forced from one cylinder through a 3*mm* bore rubber tube, the "artery," in which pulsations were obtained by the use of a small mechanical interrupter. After flowing

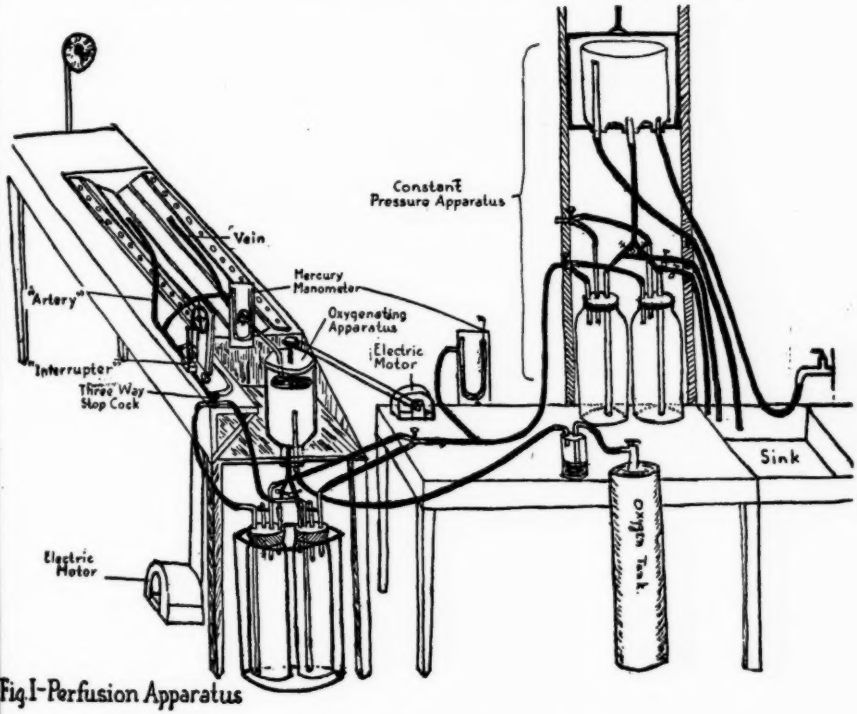



Fig. I-Perfusion Apparatus

through the dog's head, the perfusion fluid was received from the outflow canula in a 6*mm* bore rubber tube, the "vein," from which it flowed by gravity to the oxygenating chamber. The oxygenating apparatus used was essentially that described by Hooker (10). From the oxygenating chamber the blood flowed by gravity to a cylinder identical to that from which it was originally forced. By an arrangement of three-way stopcocks, the outflow cylinder could be changed, without interruption of the flow, as one cylinder emptied and the other filled. The perfusion was carried on

at a systolic pressure in the dog's artery of about 80*mm* of Hg. and a diastolic pressure of about 62*mm* of Hg.

*Operation.* Compressed air and ether through a tracheal tube were used to maintain respiration and anæsthesia. A mid-line incision was made from the lower third of the sternum to about the level of the fourth cervical spine. The skin was laid back and jugular veins exposed. Lateral side branches of the jugulars were tied off, and heavy ligatures tied around the subclavian veins and arteries. The sterno-hyoid, sternothyroid, and sternomastoid muscles were then dissected free, tied high up in the neck with a heavy ligature, to prevent oozing during the perfusion, and cut through near the sternum. The pectoral muscles were then divided, the ribs cut through, and the entire breast-plate removed, the sternal arteries and veins being tied off. The vena cava was lifted and the vertebral and azygos veins ligated. Ligatures were then placed around the left brachiocephalic artery, but were not tied until the inflow canula had been placed in the right brachiocephalic, in order to allow the left vertebral to supply the head, while the right brachiocephalic was clamped for the insertion of the inflow canula. The right brachiocephalic artery was then exposed, and ligated just distal to the point where the right carotid arises. The inflow canula was then so inserted that the tip was below the point where the artery divides into the two carotids. The inflow was then started, and about 100*cc* of perfusion fluid allowed to flow into the dog's circulation. This usually prevented any trouble from clots. The inflow was then stopped, and the outflow canula inserted in the vena cava, and the perfusion begun. The head was washed out with about 400*cc* of perfusion fluid, the outflow canula then connected with the oxygenating chamber, and the perfusion continued. This procedure necessitates a shutting off of all flow to the head during the time the outflow canula is inserted, but this is something less than a minute, and if the perfusion fluid is well oxygenated the dog continues respiratory movements uninterruptedly. A heavy ligature placed just above the heart, taking in the trachea and œsophagus, shuts off the chief point of loss of perfusion fluid, the œsophageal anastomoses. There is some slight loss through the spinal anastomoses, but this is usually temporary, and after a primary loss of about 100*cc* there is usually no further loss of perfusion fluid. Figs II. and III. show the chief ligatures and position of the canulas on the arterial and venous sides, respectively.

*Preparation of antigen.* Fresh horse serum and cows' uveal extract were used. The uveal extract was prepared by dissecting out the uveal tract of cows' eyes, and grinding with

sterile glass in a sterile mortar. It was then mixed, in the proportion 5 uveæ to 40*cc*, with salt solution, strained through sterile gauze, extracted two hours at 37° C., and after twenty-four hours in the ice-box was restrained through gauze before use. From 5 to 10*cc* of this extract were used to sensitize.

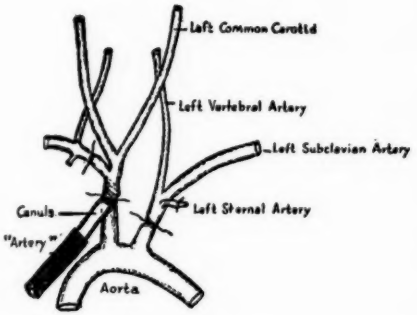



Fig II. Position of inflow Canula

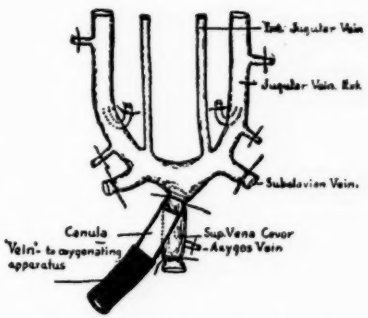



Fig. III - Position of Outflow Canula

I. *The perfusion of normal dogs with defibrinated normal blood.*

In the preliminary experiments the perfusions were continued for four to six hours. Four dogs were perfused in this series. No abnormal phenomena of any kind were observed. Beyond a moderate dilatation of the conjunctival vessels, which occurred during the first hour of the perfusion and persisted, the eyes remained entirely normal in appearance. The cornea, lens, and vitreous continued clear, and the fundus could be clearly seen. Ophthalmoscopic examination showed the retinal vessels somewhat dilated and the veins slightly engorged. No retinal hemorrhages or extravasations of blood were seen in this series, and no œdema of the eye tissues was observed either in this or subsequent series of experiments.

Histologic examination of the perfused eyes showed no lesions of any kind. In one dog, one eye was removed under ether anæsthesia before perfusion for comparison with the second eye, which was perfused for four hours. There was no demonstrable difference between the perfused and the unperfused eye.

Throughout the entire course of the perfusion the pupils remained the same size.

II. *The perfusion of normal dogs with defibrinated normal blood to which antigen (horse serum and uveal extract) was added.*

In this series of experiments, after the perfusion had run fifteen minutes and preliminary observations and measurements had been made, antigen was added to the perfusion fluid. In the two perfusions in which uveal extract was added to the perfusion fluid no difference was observed between these dogs and those perfused with defibrinated normal blood alone. In two perfusions in which horse serum was added to the perfusion fluid, the dogs showed a slight contraction of the pupil, but in all other respects the eyes remained entirely normal in appearance. The cornea, lens, and vitreous remained clear, and the fundus negative.

The contraction of the pupil in those experiments in which horse serum was added to the perfusion fluid was very slight. The maximum contraction observed was from a pupil measuring 12*mm* to one measuring 9.0*mm* in diameter. The contraction was very gradual, taking about one and one half hours before the maximum was reached. The degree of contraction seemed to be somewhat dependent upon the quantity of horse serum added to the perfusion fluid. The addition of 10*cc* gave practically no reaction, further addition of 20*cc* gave a contraction of from 1 to 2*mm*, while a third addition of 30*cc* gave a slight additional contraction. The contraction persisted throughout the entire perfusion with little tendency to redilation.

The following are protocols of normal dogs perfused with normal defibrinated blood to which horse serum was added.

EXPERIMENT IV.—*Dog 16–51* (Normal Dog). Perfusion started at 11.00 A.M. Perfusion fluid consisted of 900*cc* defibrinated normal blood and 200*cc* of Ringer's solution. Pupils equaled 12*mm* in diameter. Ophthalmoscopic examination of both fundi was negative.

*11.15* A.M. *40cc fresh horse serum added to perfusion fluid.*

*11.30* A.M. Both pupils equal 12*mm* in diameter. Fundi are negative.

*11.45* A.M. Both pupils equal 11*mm* in diameter. Fundi are negative.

*12.00* M. Right pupil equals 10*mm* in diameter. Left pupil equals 11*mm* in diameter. Fundi are negative.
*12.20* P.M. Right pupil equals 9.5*mm* in diameter. Left pupil equals 10*mm* in diameter. Fundi are negative.
*12.40* P.M. Right pupil equals 9*mm* in diameter. Left pupil equals 10*mm* in diameter. Fundi are negative.
*1.00* P.M. Both pupils equal 9*mm* in diameter. Fundi are negative.
*1.20* P.M. Both pupils equal 9*mm* in diameter. Fundi are negative.
*1.40* P.M. Both pupils equal 9*mm* in diameter. Fundi are negative.
*2.00* P.M. Both pupils still measure 9*mm* in diameter. Fundi are negative. Perfusion stopped.

EXPERIMENT V.—*Dog 16–61* (Normal Dog). Perfusion started at 11.40 A.M. Perfusion fluid 600*cc* defibrinated normal blood, 300*cc* Ringer's solution. Right pupil measures 9*mm* in diameter, left pupil measures 10*mm* in diameter. Both fundi are negative.
*12.00* M. Pupils are same diameter. Fundi are negative. *10cc horse serum added to perfusion fluid.*
*12.10* P.M. Right pupil equals 9*mm* in diameter. Left pupil equals 10*mm* in diameter. Fundi are negative. *20cc horse serum added to perfusion fluid.*
*12.30* P.M. Both pupils measure 9*mm* in diameter. Fundi are negative.
*12.50* P.M. Both pupils measure 9*mm* in diameter. Fundi are negative.
*1.10* P.M. Both pupils measure 8*mm* in diameter. Fundi are negative. *30cc horse serum added to perfusion fluid.*
*1.30* P.M. Right pupil measures 7.5*mm* in diameter. Left pupil measures 8*mm* in diameter. Fundi are negative.
*1.45* P.M. Both pupils measure 8*mm* in diameter. Fundi are negative.
*2.00* P.M. Both pupils still measure 8*mm* in diameter. Fundi are negative. Perfusion stopped.

III. *The perfusion of sensitized dogs with specific antigen (horse serum—uveal extract).*

The dogs were sensitized from three to five weeks before perfusion by intraperitoneal injection. The usual procedure was followed during perfusion, the eyes being perfused for about fifteen minutes, while preliminary observations and measurements were made, and the specific antigen then added to the perfusion fluid.

In this series three dogs were sensitized and perfused with horse serum as antigen, and three dogs sensitized and perfused with cows' uveal extract as antigen. From within a few minutes to forty minutes after the addition of the specific antigen, dependent upon the rate of flow of the perfusion fluid, a marked contraction of the pupil was observed. In all cases this primary contraction was at least twice the maximum contraction observed in normal dogs, where horse serum was added to the perfusion fluid. In various animals it averaged from 5 to 8*mm* difference in the diameter of the pupil. This primary contraction, when not extreme, was generally followed by a further gradual contraction, extending over two hours, when the maximum contraction, usually a pupil from 3 to 5*mm* in diameter, was reached. This was at times followed in turn by a gradual redilation, although the pupil as a rule never reached its original diameter. In those cases, however, in which the maximum contraction was reached at once, the pupil slowly redilated and no further contraction was observed.

The cornea, lens, and vitreous remained clear throughout. The congestion of the conjunctiva observed in the perfusion of normal dogs was vastly more marked in the dogs of this series. The eyes appeared usually extremely congested and showed a marked pericorneal injection.

The fundi in these dogs showed one very constant finding. After the eyes had been perfused with the specific antigen for about thirty minutes, small extravasations of blood appeared in the fundus. These extravasations were at first very small and lay close to the veins, near the nerve head, although several times the fundus appeared studded with them. At first they appeared as a small, discrete, reddish haze, which gradually deepened until they assumed the appearance of small hemorrhages. These increased very slowly in size and seldom attained a greater size than about one half that of the nerve head. While they lay usually close to the veins, they were as a rule definitely separated from the vessel itself, and in no case did they seem to be due to an artificial rupture of the blood-vessel. Rather did they appear to be due to a diapedesis of blood cells through the capillaries of the retina.

Histologic study of these eyes was practically negative. In some few sections small extravasations of blood were visible

upon the retina, but otherwise the eyes examined microscopically showed practically no change.

The following are typical protocols of sensitized dogs perfused with specific antigen:

EXPERIMENT VIII.—*Dog 16–8.* Sensitized on Jan. 12th, by intraperitoneal injection of 5*cc* horse serum.

*Feb. 8th.* Perfusion started at 10.50 A.M. Perfusion fluid consisted of 1100*cc* defibrinated normal dogs' blood, with 300*cc* Ringer's solution added. Head washed out with 400*cc* perfusion fluid. Both pupils equal 12*mm* in diameter. Both fundi appear entirely negative.

*11.00* A.M. *45cc horse serum added to perfusion fluid.*

*11.15* A.M. Marked pericorneal and conjunctival injection. Both pupils equal 7*mm* in diameter. The nerve head in *O. S.* appears very hyperemic, fundi being otherwise negative.

*11.30* A.M. *O. D.* Pericorneal injection more marked. Pupil equals 6*mm* in diameter. Fundus shows engorged vessels, but no hemorrhages. *O. S.* very marked pericorneal injection. Pupil equals 6*mm* in diameter. In center of nerve head are two small extravasations of blood.

*11.50* A.M. *O. D.* Marked congestion. Pupil equals 8*mm*. Fundus negative. *O. S.* Extreme pericorneal injection. Pupil equals 5*mm* in diameter. Vessels of fundus very full, and extravasations of blood on nerve head are slightly larger.

*12.20* P.M. *O. D.* Pupil equals 7*mm* in diameter. External appearance of eye unchanged. Fundus unchanged. *O. S.* Pupil equals 4.5*mm*. Fundus appears as before described. External congestion of the eye is extreme.

*12.45* P.M. *O. D.* Extreme pericorneal injection. Pupil equals 6*mm* in diameter. Fundus is engorged and hyperemic. *O. S.* Pupil equals 4.5*mm* in diameter. The extravasations of blood noted before appear about the same. External appearance of the eye is as before.

*1.20* P.M. There is no apparent change in appearance of the eyes over the last note. *O. D.* Pupil equals 6*mm*. *O. S.* Pupil equals 4.5*mm*.

*1.45* P.M. *O. D.* Pupil equals 5*mm* in diameter. External appearance unchanged. The fundus is engorged, and the nerve head appears very hyperemic. *O. S.* Pupil equals bare 3*mm* in diameter. The pericorneal injection is extreme. The extravasations of blood on the nerve head now have the appearance of well-formed hemorrhages.

*1.55* P.M. The eyes show the same external appearance before described. The fundi show no further change. The

pupils have begun to redilate slightly. *O. D.* Pupil equals 7*mm* in diameter. *O. S.* Pupil equals 5*mm* in diameter. Perfusion stopped at 1.05 P.M.

The eyes were preserved in 4 per cent. formalin, and later examined microscopically. Both eyes appear normal, with the exception of a small hemorrhage in the retina near the nerve head in the left eye. There is no œdema of the tissues.

EXPERIMENT IX.—*Dog 15–73.* Sensitized on Nov. 15th, by intraperitoneal injection of 5*cc* of horse serum.

*Jan. 7th.* Perfusion started at 11 A.M. Pupils measure 12*mm* in diameter. Fundi are negative to ophthalmoscopic examination.

*11.10* A.M. Pupils measure 12*mm* in diameter; fundi appear negative. 30*cc* of horse serum added to perfusion fluid.

*11.30* A.M. Pupils measure 4*mm* in diameter. *Fundus O. D.* Vessels are engorged. One small extravasation of blood is seen near upper nasal vein, fundus otherwise negative. *Fundus O. S.* Two small hemorrhages are seen in upper part of field, otherwise examination is negative.

*2.00* P.M. There has been a gradual redilation of the pupils until they now measure 8*mm* in diameter. At 1 P.M. 20*cc* horse serum was again added to the perfusion fluid, no further contraction being noted. The hemorrhages have become more sharply defined. No new extravasations of blood have appeared. Perfusion stopped.

EXPERIMENT XI.—*Dog 16–9.* Sensitized by intraperitoneal injection of 5*cc* of cows' uveal extract on Jan. 12th.

*Feb. 16th.* Perfusion started at 11.30 A.M., through right carotid alone; no perfusion through left eye. Pupils both measure 14*mm* in diameter. Fundi negative.

*11.50* A.M. Eyes are unchanged in appearance, *35cc cows' uveal extract added to perfusion fluid.*

*11.55* A.M. Right pupil has contracted to 6*mm* in diameter. Left pupil is unchanged in size. Fundus of right eye is engorged, but shows no hemorrhages.

*12.05* P.M. Right pupil has redilated to 9*mm*, fundus shows no further change. Left eye unchanged.

*12.30* P.M. Eyes show no further change.

*1.00* P.M. Both eyes are as before noted. Fundi are negative.

*1.30* P.M. Right pupil measures 9*mm* in diameter, pericorneal injection more marked, fundus is negative. Left eye unchanged.

*2.15* P.M. There has been no change since last note.

*3.10* P.M. Right pupil equals 10*mm* in diameter. There

is marked pericorneal injection. Ophthalmoscopic examination is negative. Left eye is unchanged. Perfusion stopped.

EXPERIMENT XII.—*Dog 16–10.* Sensitized on Jan. 12th, by intraperitoneal injection of *7cc* of cows' uveal extract.

*Feb. 18th.* Perfusion started at 11 A.M. Pupils measure *12mm* in diameter. Fundi are negative to ophthalmoscopic examination.

*11.10* A.M. Pupils are *12mm* in diameter, fundi are negative. *50cc cows' uveal extract added to perfusion fluid.*

*11.20* A.M. *O. D.* Pupil measures *8mm* in diameter. Fundus shows a small number of small ill-defined extravasations of blood. *O. S.* pupil equals *9mm* in diameter. There are many small ill-defined hemorrhages scattered diffusely throughout fundus.

*11.40* A.M. Pupils of both eyes measure *8mm* in diameter. The fundi are unchanged since last note.

*12.00* M. Pupils are *8mm* in diameter. The hemorrhages throughout the fundi are more clearly defined. The pericorneal injection is marked.

*12.30* P.M. The pericorneal injection is somewhat more intense and the hemorrhages in the fundus somewhat better defined, otherwise the condition of eyes is unchanged.

*1.10* P.M. The condition of the eyes is unchanged, except that the pupils both measure *7mm* in diameter.

*1.45* P.M. Pupils measure *7mm* in diameter. Pericorneal injection is marked. The hemorrhages in the fundi are well defined.

*2.15* P.M. Both pupils measure *6mm* in diameter, fundi and general appearance of eyes unchanged.

*2.45* P.M. The condition of the eyes is unchanged since the last note. Perfusion stopped.

## DISCUSSION.

Schultz (11), in 1912, showed that horse serum could stimulate the smooth muscle of the normal cat to contraction, and that the contraction was independent of nervous control. The slight contraction of the pupil of the normal dog perfused with horse serum added to the perfusion fluid is in accord with this observation. Both the dilator and constrictor fibers of the iris are of smooth muscle, the constrictor fibers being by far the stronger and more numerous. While the slight contraction of the pupil in the normal dog perfused with horse serum is

readily explicable by this observation of Schultz, the contraction of the pupil of the sensitized dog perfused with specific antigen was vastly greater than that of the normal dog, and no contraction of the pupil whatsoever was observed in the normal dog perfused with uveal extract, while the sensitized dog gave a marked reaction. Furthermore, the extravasations of blood in the retina were observed only in sensitized dogs perfused with the specific antigen. It seems safe, therefore, to conclude that the reactions observed in the sensitized dogs were truly of an anaphylactic nature.

The contraction of the pupil of the sensitized dogs perfused with the specific antigen is in direct accord with the observations of Dale (12), who observed the contraction of the sensitized uterus suspended in specific antigen; of Schultz (13), who observed the contraction of sensitized smooth muscle when exposed to specific antigen and of Szily (9), who observed a contraction of the pupil upon the intravenous injection of specific antigen in rabbits previously sensitized by intraocular injection.

These experiments indicate that this contraction of the pupil is due to a sensitization of the tissues of the iris, which contract, as does other sensitized smooth muscle, in the presence of specific antigen. The possibility of it being due to a humoral reaction seems rather remote. In preliminary experiments it was found that about 300*cc* of salt solution were sufficient to wash out the head, and in all experiments upon sensitized animals the head was washed out with approximately 400*cc* of perfusion fluid, which was allowed to escape. As there was no œdema of the ocular tissues whatsoever, it seems probable that all of the dogs' own blood was washed out by this means. The promptness of the contraction of the pupil and the apparent lack of any interchange between the aqueous humor and the ocular circulation make it unlikely that the body, necessary to cause the anaphylactic reaction, could have been furnished by the aqueous humor, although this is a possibility which cannot be excluded.

The seat of the reaction in this case seems therefore to be in the cells of the iris itself, which probably are the seat of the sensitization needed to complete the anaphylactic reaction. This observation seems to be another instance of cellular

anaphylaxis and is in accord with the well-known experiments of Pearce and Eisenbrey (14), Schultz (13), Dale(12),Weil(15), and Coca (16).

No explanation can be offered at present for the extravasations of blood in the retina of the anaphylactic dogs. Hemorrhages have been observed over the peritoneum and in other parts of the body in animals that have recovered from anaphylactic shock, but the mechanism of these hemorrhages is not clear. From the character and mode of formation of the extravasations of blood observed in these experiments, it seems probable that the endothelium of the capillaries is in some way altered, in the anaphylactic state, so as to allow the escape of red cells by diapedesis.

## SUMMARY.

The smooth muscle of the iris of normal dogs contracts slightly when perfused with defibrinated normal dogs' blood to which horse serum has been added, and the amount of contraction seems directly proportional to the amount of horse serum in the perfusion fluid.

Sensitized dogs, the heads of which are perfused with specific antigen, show a definite ocular reaction which is anaphylactic in origin. The reaction consists (1) of a primary contraction of the pupil, greater than that in the normal animal receiving serum, and followed usually by a further gradual contraction, and (2) of small extravasations of blood throughout the fundus. The contraction of the pupil is similar to that observed in sensitized smooth muscle in the presence of specific antigen, and is an evidence of cellular anaphylaxis occurring *in vivo*. No definite explanation can be offered for the extravasations of blood in the fundus, beyond the fact that they are somewhat analogous to the hemorrhages observed elsewhere in animals recovering from anaphylactic shock.

## CONCLUSIONS.

I. The eye can be sensitized by intraperitoneal injection, and anaphylactic phenomena can be elicited in it by antigen carried by the blood stream.

II. The anaphylactic phenomena consist in a primary contraction of the pupil, followed by a further gradual contraction, and in small extravasations of blood throughout the fundus.

III. The contraction of the pupil seems to be due to a reaction of the smooth muscle of the iris to the specific antigen in the perfusion fluid, and is in definite accord with the cellular theory of anaphylaxis.

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# A STUDY AS TO WEIGHT AND PERCENTAGE OF SOLIDS OF CATARACTOUS LENSES.

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ONE finds in various publications the statement that in cataractous lenses the amount of insoluble albuminoid is increased. This work was started primarily to discover whether these statements were founded upon fact or theory but owing to certain difficulties encountered the amount of insoluble albuminoid was not determined (except in one or two cases) but as the weight and percentage of solids was determined and as this showed a rather wide variation I have deemed it of sufficient importance to place on record.

The lenses showed all manner of variations seen in general run of work, from removal in capsule to soft lenses with some loss of cortical substance, from immature cataracts to lenses showing calcareous degenerations.

It is unfortunate that this work was not done in all cases upon lenses extracted in capsule, as there necessarily arises some error in both weight of lens and also percentage of solids due to some cortical substance being lost, but in Baltimore material of that character is very rare.

The lenses after extraction were immediately placed in a ground glass stoppered weighing tube and weighed within a few hours, then placed in a vacuum and dried to constant weight.

The heaviest was Mary M. (No. 8), a colored woman of eighty, with an amber cataract, being as you see by the table 0.2418 Gm., while the lightest was Mrs. P. (No. 19), the last one in the table, 0.0988 Gm., but this consisted of nucleus chiefly, the soft substance being lost; but the right lens only

weighed 0.1154 Gm., so that these were apparently small lenses.

The average weight of 0.1640 Gm. corresponds very closely to No. 1, which was removed in capsule (0.1604 Gm.).

By the study of their weight according to age one finds that those of eighty or over showed an average weight of 0.1968 Gm., and also a slight increase in percentage of solids, 29.31.

In contradistinction to this, those under sixty show an average weight of 0.1335, but owing to one case of amber cataract with a very high percentage of solids, No. 16, the average percentage of solids was 27.36, being 0.34 greater than the general average.

In 1913, ARCHIVES OF OPHTHALMOLOGY, Vol. XLII., I showed that the weight of an infant's lens (43 days old) averaged 0.0953 Gm., so one sees here an adult lens, being 1.7 times the size of the infant's. Much to one's surprise the percentage of solids in the infant's lenses showed a slight increase over those of senile cataracts, 27.57 in comparison to 27.02, but this variation is easily within the limits of error.

Sappey's estimation of 0.218 Gm. for an adult lens seems to be a little high in view of my findings, as one could hardly believe that opacification would lessen the weight materially, and while some cortex was lost in some of the cases, No. 1 was removed in capsule and as you see corresponds very closely to the average weight. Priestley Smith and J. Treacher Collins place the percentage of solids in adults at 29. Here again my findings would tend to place them somewhat below that figure (27.02).

I hope some of my colleagues who have access to many lenses removed in capsule will either verify these figures or give us ones still more accurate.

TABLE I.

| No. | Name | Age | Eye | Condition of Lens | Wt. Lens | Solids | Percentage of Solids |
|---|---|---|---|---|---|---|---|
| 1 | Mr. Abrams..... | 80 | O.D. | In capsule. | 0.1604 | 0.059 | 36.78 |
| 2 | Mrs. Brice..... | 78 | O.D. | Large amount soft cortex. | 0.1342 | 0.0162 | 12.04 |
| 3 | Mrs. Valentine. | 66 | O.S. | Small amount soft substance. | 0.1690 | 0.0404 | 24.50 |
| 4 | Mr. Donlon.... | 50 | O.S. | Calcareous deposits on ant. capsule. | 0.1342 | 0.0273 | 21.35 |
| 5 | Mrs. S. Dorsey. | 60 | O.D. | Large nucleus, little soft substance. | 0.1762 | 0.0534 | 30.23 |
| 6 | Mrs. Hull..... | 74 | O.D. | Little soft substance. | 0.1613 | 0.0692 | 42.96 |
| 7 | Mrs. Brown.... | 75 | O.D. | Large amount soft substance. | 0.0624 | 0.0283 | 45.35 |
| 8 | Mary M..... | 80 | O.S. | Amber. | 0.2418 | 0.0778 | 32.18 |
| 9 | Mrs. Price..... | 54 | O.D. | Considerable soft substance. | 0.1154 | 0.0273 | 23.66 |
| 10 | Mr. Irvin..... | 67 | O.D. | Amber cataract. | 0.1948 | 0.0685 | 35.11 |
| 11 | Mrs. Kilduff... | 59 | O.S. | Hypermature. | 0.1529 | 0.0456 | 26.68 |
| 12 | Mrs. Kozlonsky | 61 | O.S. | Mature. | 0.1631 | 0.0460 | 28.20 |
| 13 | Mr. L. Stein... | 61 | O.S. | Large amount of soft substance. | 0.1253 | 0.0282 | 22.58 |
| 14 | Mr. B..... | 80 | O.S. | Large amount of soft substance. | 0.2090 | 0.0377 | 18.04 |
| 15 | Mrs. Gains..... | 74 | O.D. | Amber. | 0.1810 | 0.0446 | 24.64 |
| 16 | Mr. Oppety.... | 50 | O.D. | Amber. | 0.1661 | 0.0598 | 36.00 |
| 17 | Mrs. Radford.. | 63 | O.S. | Cat. Niger. | 0.1988 | 0.0628 | 31.58 |
| 18 | Miss Moore.... | 60 | O.S. | Small amount of soft substance. | 0.1569 | 0.0444 | 28.30 |
| 19 | Mrs. Price..... | 55 | O.S. | Some cortex lost. | 0.0988 | 0.0288 | 29.15 |
| | Average | 66 | | Average | 0.1640 | 0.0451 | 27.02 |

# CAN THE DEVELOPMENT OF MYOPIA BE ARRESTED AND ITS DEGENERATIVE CHANGES BE PREVENTED?

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(Translated from the *Archiv f. Augenhlk.*, Vol. LXXIX., 1915.)

AS the question contained in the above title is still open, I have thought it worth while to collect material from the out-patient department of the Zurich University Eye Clinic and my private practice in order to see whether an examination of these cases will throw any further light upon this question. The examination has been particularly directed to cases of anisometropia and to myopes who have been wearing full correction; these cases have been selected from the case-histories of the last ten years.

Gamper, at my suggestion, has measured carefully the orbital index of anisometropes of high degree (a difference of refraction in the two eyes of from 10 to 25 D.) by means of radiographs. The contours of the orbital entrance could not be recognized in the plate, but the orbit itself was well defined. A study of these plates showed that the size of the orbit much more than the orbital entrance depended upon the development of the various accessory cavities. These cavities vary to such an extent that a relationship between them and the orbit must exist. Stereoscopic radiographs gave us no more information than the ordinary photograph. These orbital measurements, made with carefully adjusted radiographs which were very much more exact than those published by Stilling and other authors, showed me that there was no close relationship between the contents of the orbit on one side and the refraction

of the eye of the same side; and, therefore, that there is no justification for Stilling's theory.

In addition to the question of full correction to which I shall return later, I was able to collect 50 anisometropes in whom the refractive difference consisted in at least 10 D. The case-histories of these 50 anisometropes resemble the 100 cases published by Gamper so closely that I can report upon a total of 150 cases as follows:

1. The refraction in one eye:

| | | | |
|---|---|---|---|
| In 49 cases | | −10 to | −15 D. |
| 45 | " | −15 " | −20 D. |
| 37 | " | −20 " | −25 D. |
| 19 | " | −25 " | −30 D. |

In the other eye:

| | | | |
|---|---|---|---|
| In 52 cases | | −1.0 to | −10.0 D. |
| 51 | " | +1.0 " | + 5.0 D. |
| 47 | " | Emmetropia. | |

2. The position of the eyes in 57 cases was divergent; in 9 convergent; and in 5 nystagmus was present.

3. An examination of the refracting media showed more or less dense maculæ of the corneæ, in one eye in 63, in both eyes in 15, and consequently together in 78 eyes. Furthermore, in 1 case there was radiating opacity in the lens, in 3 a posterior polar cataract, in 4 a cerulean cataract, in 1 a central cataract, in 1 an incipient cataract, and in 1 a fine pupillary membrane. In 29 there were marked vitreous opacities, and in 1 case this was present in both eyes. The fundus examination revealed a myopic eye-ground in 128 cases; in 120 of these in the near-sighted eye and in 8 in both eyes. Sixty-eight cases showed a narrow conus; in 39 there were peripapillary atrophies and disease of the macula. In 10 the macular spot of Fuchs was present; there were hemorrhages in the macula in 5 eyes, and staphyloma verum in 6.

Considering the difference in vision between the two eyes, the strabismus, and the difference in refraction, these 150 anisometropes have practically only used one eye. In 10 cases a fair amount of vision with a correcting glass was

possible in both eyes, but owing to the refractive difference of 10 to 20 D. the two eyes could not be used together. Though these 150 very weak-sighted eyes were not used, they gradually became more and more myopic and in many cases the myopia became excessive. With this, the well-known myopic eye-ground changes occurred not only in the form of crescents and ring atrophies about the disk, but of diseases of the macula, retinal hemorrhages, and opacities of the vitreous. These observations show definitely that the use of the eye has nothing to do with the degree or the deleterious consequences of a near-sighted eye.

Steiger in his book on the development of spherical refraction of the human eye has shown the error of our views on so-called myopia and on those factors which are believed to develop near-sightedness, as well as the uselessness of measures which are believed to arrest the progress of the myopia. In my opinion no cases are more suited to support this view than these 150 anisometropic eyes. I do not think that restricted use of the eyes can in any way prevent the progress of myopia, nor prevent the deleterious consequences.

The cause for the refractive difference in these anisometropic eyes consists probably in heredity, just as it does in myopia. The near-sightedness was probably in these cases inherited in one eye and later developed the degree and the accompanying fundus changes which had previously been determined upon at the time of conception and were independent of the use or the non-use of the eye in question.

On the other hand, a careful inquiry among these 150 cases gave results which were not exactly favorable to the law of inheritance, for among these 150 patients I was able to obtain information only in 18 that myopia existed either in the parents, grandparents, or relatives. Small degrees of ametropia are, however, often not recognized by the patients, and if one eye is normal the defect of the other is ignored.

It is of interest to ask whether the degree of myopia as well as its deleterious sequelæ are both inherited tendencies. Our experiences with myopic eyes have taught us that with increasing myopia changes in the eye-grounds of progressive development occur. The abnormal lack of resistance and distension of the posterior scleral segment are the cause for

the atrophy of the retina and choroid about the posterior pole of the eye, presumably the result of a mechanical injury and consecutive distension. In high-grade myopia there is a congenital lack of resistance on the part of the sclera. In the medium and low degrees of nearsightedness the conditions are somewhat different. In these, typical myopic changes in the eye-grounds may be present. In myopes under 10 D. there may be more or less marked changes in the retina and choroid which in no way differ from those observed in excessive myopia. These cases are not nearly so rare as is generally believed. I have found in about 4000 myopic patients, 218 diseases of the macula, and of these, 49 eyes with a myopia of less than 10 D. showed disease of the yellow spot. In 67 there was a temporal crescent or a ring atrophy. Generally the changes were bilateral and frequently more pronounced on one side than the other. The pronounced fundus disease corresponded not so much to the degree of the myopia as to the poor vision obtained.

Haab has drawn attention to this interesting condition, and in 1890 at his suggestion Schweizer published a dissertation on the deleterious sequelæ of the varying grades of myopia. This study was based upon 5000 myopic eyes obtained from the private journals of Professor Haab. In 265 eyes the macula was diseased, and among these in 44 the myopia was under 10 D. This corresponds to the figures which I found. Schweizer draws attention to the prognostically unfavorable conditions present in the form of maculæ corneæ and astigmatism. In these 44 cases with macular diseases in eyes under 10 D., these complications were present in 36. In the examination of my patients I was struck with the frequent presence of maculæ corneæ. In the 150 cases of anisometropes, in 78 eyes these corneal changes were present and in the study of all the other cases of myopia maculæ corneæ occupied an important rôle. In 4000 myopes maculæ corneæ were present in 187.

Steiger,[1] in an article in which he gives the result of an examination of school children and comes to the conclusion that the opacity of the cornea can not be regarded as the cause for the myopia. Though this examination in various directions is of great value, I do not think that it is quite capable of

[1] Steiger, *Zeitsch. f. Augenhlk.*, vol. xxv., 1911.

solving this question. Steiger examined school children between the 7th and 12th year; a number of myopes, however, do not develop until after the 12th year and show progression after that year. Furthermore, I know of a series of patients who became nearsighted during an observation period of a number of years following interstitial or eczematous keratitis or after corneal opacities from injuries. These cases were not adolescents but were often in the 20's or 30's. Furthermore, cases of congenital cataract and zonular cataract frequently develop myopia in the course of years.

Korsak[1] found in 50 patients with zonular cataract in the Zurich Eye Clinic 10 myopes, while in my 12 zonular cataract patients 5 were myopic.

I am inclined to agree with Meyerhof and other authors, who assume that the corneal opacity is the cause for the development of the myopia, or at least believe that there is an association between the opacity of the cornea or of the lens and the myopia. In these patients which I have just mentioned, heredity of myopia was present in only one.

We must consider heredity as the most frequent etiological factor in the development of myopia, though an acquired myopia even if it plays but a subordinate rôle, can not be entirely disregarded. We do not understand how the development of myopia occurs in the cases with disease of the cornea and of the lens. At the same time, I know some cases who were myopic even before the onset of the corneal changes and later became more nearsighted. It is possible in these cases to consider that heredity and the maculæ corneæ together were unfavorable factors. These unfavorable accessory conditions in myopia, particularly the maculæ corneæ, were particularly well represented in the 150 cases of anisometropia, in whom 78, that is more than half, presented opacities of the cornea. This high percentage can not be regarded as accidental, and there must be a certain causative relation between the corneal opacity and the myopia. The question naturally presents itself, whether the nearsightedness and the changes in the fundus in these weak eyes have not increased on account of the great functional incapacity. If this is correct, the development of myopia may be regarded as a degenerative process,

[1] Korsak, *Zurich Dissertation*, 1914.

and a number of factors are in favor of this view. Steiger (*l.c.*) speaks of a degenerative process in excessive myopia, and he is inclined to accept one. To solve this question, no better cases can be found than those of marked anisometropia, and particularly those occurring in young individuals of ten and under, with one normal eye and the other myopic eye of 20 and more D., with extensive fundus changes. These cases are of great interest and show that the excessive forms of myopia and its deleterious sequelæ may develop without any use of the eye, and furthermore show that the retina and choroid probably in these cases rapidly degenerate on account of this early defective or nearly complete lack of function. Similar conditions are found in general medicine. Muscles or nerves rapidly degenerate when there is loss of function. Albinotic eyes which present congenital cataract, nystagmus, and other signs of degeneration are often myopic. It is possible that functional incapacity may lead to a degeneration of all the ocular tunics, not only of the choroid and retina, but particularly of the sclera, with changes in the vessels which in turn produce a lack of resistance and a tendency to distension.

If we accept this possibility, we find that it must apply for the anisometropic eyes which are not used for seeing and particularly for the degeneration of the retina, as it occurs in excessive myopia. It is not probable that anatomic changes in macular disease of a myopic eye which has been excessively used differ from the same changes of the fundus occurring in anisometropic and unused nearsighted eyes. At least the eye-ground pictures in both of these cases are similar. In every excessively myopic eye, vision sinks below a certain level and the eye suffers from a more or less marked functional incapacity. This in turn can be the cause for the degenerative myopic process. At the same time, in most cases a congenitally abnormal lack of resistance of the posterior scleral segment causes the myopic changes.

What is the result of our treatment? I was probably one of the first in 1906 to draw attention to the bad results which followed the operation for myopia, and to the fact that the results which at first were good, after a brief period of time became rapidly worse. At that time I examined 75 cases which had been carefully operated upon by Professor Haab.

The results now that eight additional years have elapsed are even worse than they were before. Of these 75 eyes, I was able to examine again only 50. The vision became worse, principally from diseases of the macula, retinal hemorrhages, and detachment of the retina. In my previous statistics of these 75 aphakic eyes, 11 showed disease of the macula, retinal hemorrhages occurred in 9, and retinal detachment in 8. Of the 50 cases which have just been re-examined, we found disease of the macula in 18, retinal hemorrhages in 11, and detachment of the retina in 13. Corresponding to these changes, vision has become correspondingly worse or completely lost. Our hopes based upon the success of phakolysis have not been fulfilled, and this operation can in no way be regarded as a therapeutic measure for myopia, as the myopia does not remain stationary after the operation, nor are the severe sequelæ prevented.

If we now examine the eyes of excessive myopia which have not been operated upon, we may use of these same 50 eyes the cases where the operation was performed in only one eye. In addition I have selected 50 more patients with excessive myopia; in other words, a total of 150 eyes. These patients all showed a myopia of at least 15, an average of 18 D., and were all examined for the first time after their 20th year, a period when they had reached full development. The length of observation varied from 10 to 20 years, an average of 13. Of these 150 eyes, vision in only 31 remained the same, and in those in which the period of observation, from 10 to 12 years, was the shortest. In the other 119 eyes the diminution of sight varied from 0.1 to 0 on account of disease of the macula, retinal hemorrhages, vitreous opacities, and detachment of the retina.

If we compare the visual results of the 50 non-operated eyes of the one-sided aphakic patients with the 50 patients who were not operated upon, we find that the vision of the former series of 50 eyes, though the eyes were subjected to greater work, as the aphakic eye is usually used only for distance, the myopia in the former cases remained more stationary than the vision of the 50 patients who were not operated upon. This confirms what I have stated in the discussion of the anisometropic eyes, that the progress of the myopia and the diminu-

tion of vision with or without changes in the fundus can not be made dependent upon the amount of work which the eye is subjected to.

One almost gets the impression that use of the myopic eye is possibly more favorable than non-use. This is in direct contradiction to the generally accepted belief. At the same time, it confirms the statement previously made, that the myopic process is a degenerative process, which has nothing to do with the amount of use which the eye is subjected to. It must be acknowledged that for operation the eye which was the most nearsighted was the one selected for operation. These more myopic eyes did not show more extensive changes in the eye-grounds, especially as eyes with extensive changes in the eye-grounds were never operated upon by Professor Haab.

Another striking feature was found on examining these 150 eyes for the degree of myopia. In 68 the myopia increased from 1 to 6 D., notwithstanding that all the patients had reached full growth at the time of the first examination. In 82 the myopia remained stationary. With the increase of myopia the diminution of sight was not always proportional.

We have also been able to form some conclusions upon the value of general treatment. A number of our patients were those connected with the university, with the high schools, with the teachers' college, etc., and who came for consultation on account of their high myopia with either a normal retina or beginning disease of the macula, and in certain cases with central retinal hemorrhages. These patients are particularly valuable for our statistics because they were intelligent and followed for years our directions with remarkable energy and pertinacity. Some, in fact, changed their profession. Though occasionally an apparent improvement was observed, on the whole we must acknowledge that both the sight and the disease of the retina slowly progressed with or without an increase in the myopia, and frequently the other and the better eye after a number of years developed the same macular disturbance. In one of these patients whom I saw for the first time ten years ago, there was a large round atrophic macular area in the right eye. As the patient stated that he had never been able to see much with this eye, I was inclined to regard this as a congeni-

tal coloboma of the macula. The myopia in that eye was 16 D. and in the left 12 D. In the latter eye with this glass the patient had normal vision and the fundus seemed absolutely normal. As the patient was an overseer of a machine shop, a profession which required very good sight, I advised him to favor this eye as much as possible. He therefore gave up his position, no longer used his eye even for reading, but notwithstanding, two years ago a small atrophic area appeared near the left macula which gradually enlarged until it had reached the same size as the atrophy in the right eye. A parallel of this case are the macular diseases with or without hemorrhages. These come and go without in any way being influenced by the amount of work which the eye is subjected to. On asking a number of these intelligent patients for their opinion on the value of this general treatment, I received from nearly all of them the answer that the course of the eye condition was apparently unchanged. It is, of course, true that small macular affections and retinal hemorrhages in delicate anæmic individuals are favorably influenced by general tonic treatment: iron and arsenic, and particularly subcutaneous injections of caccodylate of soda in addition to the local treatment (subconjunctival salt injections, leeches, etc.).

Other and similar cases convinced me that the usual directions which we give these myopic patients have but little effect. In my experience the strongly myopic eyes which have been subjected to a great deal of work undergo the same change as the anisometropic amblyopic eyes which are practically not used. In other words, myopia gradually increases to that degree and to that amount of vision, irrespective of external influences, which had been predecided in the "Keimesanlage."

Very severe complications, as is known, occur even in the lowest degrees of nearsightedness, nor can we decide whether the myopia is going to remain stationary or will later on develop. It is impossible to state the degree at which the degenerative character of the lesion begins and the point at which a myopic eye begins to be regarded as a degenerated eye. The progeny of a weak myope may develop severe forms of myopia and it seems that every myopic eye carries with it the germ of degeneration. In other words, our treat-

ment of myopia has not fulfilled the hopes that have been placed upon it.

The question of the full correction is worthy of study. I have examined 350 eyes who have all been patients at our clinic and in my own private clientele. In order to determine what influence the full correction has upon progress of myopia, only youthful individuals were selected. It was found that the myopia increases most in the 6th to the 10th year, on an average of 2.5 D. during an observation period of 7½ years. From the 11th to the 15th year the increase in myopia was only 0.8 D. during a controlled period of 6 years. In the 16th year, with an observation period of 5 years, the increase consisted in about 2 D. From the 17th year on, the tendency to increase of myopia gradually diminished without entirely ceasing, and in certain cases an increase was observed even in the 25th, 30th, and 35th year.

These statistics show that after the 10th year the myopia on an average increases just as much as it did before this year. I am therefore inclined to regard the statistics of Steiger, which are based upon an examination of school children with maculæ corneæ from 7 to 12 years of age, as not being suited to decide the question as to whether corneal opacities are the cause for the myopia.

Of the 175 myopic patients who wore glasses and who were examined on an average after 6 years, 134 were fully corrected, and the remainder were slightly under-corrected (1–2D.) or strongly under-corrected (2 or more D.). Of the 134 fully corrected cases, the myopia remained stationary in 39.5%, while in 34.3% of the slightly under-corrected the myopia progressed and in 26.2% of the strongly under-corrected the myopia also progressed.

In 30 of the weakly under-corrected cases (73.3%) and in 11 of the strongly under-corrected cases (63.6%) the myopia remained stationary. Though the 41 under-corrected cases are only a fraction of the 134 fully-corrected cases, the figures are nevertheless sufficient to show that the myopia may remain stationary just as well with the under-correction as with the full correction. We must further acknowledge the sad experience that full correction is not always capable of preventing the myopia from progressing.

The increase of myopia and its deleterious consequences can not be prevented by methods which are at our command. We agree with Steiger, that independent of external factors, the degree of myopia depends upon the congenital "factors." My observations are based on the examination of 150 anisometropes of high grade, of 4000 myopic eyes of various grades, of 150 excessive myopes, of 62 patients with zonular cataract, of 50 patients who had been operated upon with phakolysis and who had been observed for a period of 16 years, and finally of 134 fully-corrected myopes. These have shown that a restriction of close work, hygiene of the eyes, phakolysis, selection of profession, correcting glasses, must be regarded as of but little avail in favorably influencing the progress of myopia and that myopia must be regarded as a congenital evil which can only be influenced by the proper selection of individuals for marriage.

As changes in the vitreous and in the eye-grounds in excessive myopia must be regarded as morbid and degenerative conditions, these eyes must be favored, though I could not convince myself that much was obtained by following out any particular line of treatment, and I soon became convinced that we could change little in the favorable or unfavorable course of a myopia as this is probably decided upon during the developmental period of the individual. The practical results of this investigation are as follows:

It is not necessary to be too strict in the choice of a profession. A myope may become an engineer just as well as a farmer or a forester; and that school work, close work, poor illumination, improper desks, etc., do not necessarily increase the myopia, but that in most cases it is governed entirely by factors which can not be influenced by external conditions. This means that children that are otherwise normal should not necessarily be restricted in their work. At the same time nervous, delicate, or overburdened children should not be permitted to take up side studies even if the myopia will thereby not be influenced. Furthermore, full correcting glasses which are ordered with the idea of controlling the progressiveness of the myopia need not be prescribed if they cause disturbances, for even weaker glasses may obtain the same results and are often more comfortably worn. On the other hand, we have seen no bad

effect from the full correction and shall continue to use this in future as long as it causes no annoyance to the patient. As for the prescribing of a distance and a near glass, we should be guided principally by the patient himself. If the full correction can be worn for close work with comfort, it should be prescribed, otherwise in low degrees of myopia the patient may dispense with glasses or may use a weaker glass than the one used for distance. In other words, in prescribing glasses we should be guided by the wants of the individual patient and rather more by his subjective symptoms than has been previously the general habit.

# REPORT OF THE PROCEEDINGS OF THE SECTION ON OPHTHALMOLOGY OF THE NEW YORK ACADEMY OF MEDICINE.

By LEWIS W. CRIGLER, M.D., Secretary.

MONDAY EVENING, MAY 15, 1916. GEORGE H. BELL, M.D., CHAIRMAN.

Dr. Martin Cohen presented a patient upon whose right eye he had performed an **optico-ciliary neurectomy for painful absolute glaucoma.** The case was that of a woman seventy years of age who had had trachoma in the right eye only. Symptoms of glaucoma developed in this eye about eight months ago. When seen by Dr. Cohen six weeks ago she presented the typical picture of absolute glaucoma in the right eye only, the left being apparently normal. Iridectomy, preceded by posterior sclerotomy, was performed. All symptoms were relieved temporarily, but at the end of four weeks the tension had risen to 60*mm* of mercury and the pain became excruciating. Enucleation was then advised, but the patient refused. She readily gave her consent, however, to the less radical procedure. Schirmer's method was followed in performing the operation. 5*mm* of the optic nerve was resected. The eyeball has not been painful since the operation. The cornea is anæsthetic and the tension is now 25*mm* of mercury. The patient has had an ophthalmoplegia externa since the operation two and a half weeks ago. This may or may not be permanent, depending on whether or not the extraocular nerves were severed at the time of the operation.

Discussion: Dr. Claiborne said that he had seen De Wecker perform a similar operation thirty years ago for the relief of sympathetic ophthalmia. He does not approve of the operation.

Dr. SCHOENBERG said that he had studied a case that had been operated on by Dr. Schirmer. He said that the tension had not been relieved but that the pain had, and inasmuch as that was the main object desired, he thought the operation quite justifiable.

Dr. LAMBERT asked if operation had obviated the necessity of an enucleation at some later period. Dr. COHEN said that he thought it had.

Dr. T. H. CURTIN showed a case of **buphthalmos with favorable results following trephining.**

The case was that of a child six months of age, with a history of increasing enlargement of both eyes since birth. When first seen both corneæ were opaque, sclera and corneæ quite thin, and the tension very much increased. The child was apparently blind. His general condition was good. An Elliott trephining operation was performed on each eye, and in two weeks both corneæ were more transparent. Although the tension was considerably reduced, it was decided that both eyes should again be trephined to make sure that there should be sufficient drainage from the interior of the eye to control the tension. This was done three weeks after the date of the first operation.

At the time of this report, ten months since the date of the last operations, both eyeballs are reduced in size but still larger than normal. The tension is now normal in each eye.

The patient now sixteen months old can see sufficiently well to find his way around the ward alone. Vision is confined almost entirely to the right eye, as the opacities in the left cornea are much more marked than in the right.

DISCUSSION: Dr. VIRDEN stated that he had had a case in which he made his trephine openings back of the ciliary body. He succeeded in reducing the tension in both eyes as well as the size of the eyeballs. Vision was still reduced to perception of light.

Dr. WOOTTON said that he had performed the operation but not with much success.

Dr. MARPLE thought that the corneal opacities in Dr. Curtin's case must not have been very deep to have cleared up so well.

Dr. MARPLE showed a case of **epibulbar epithelioma.** The

growth was of four weeks' duration. A small piece of the tumor was excised for microscopic examination which revealed the nature of the growth. In view of its intimate adherence to the sclera, Dr. Marple thought it best to try the use of radium before resorting to more radical measures.

Dr. WOOTTON wanted to know the chances of metastases, to which Dr. Marple replied: very slight.

Dr. WEIDLER said that he had seen a case very similar in appearance to Dr. Marple's, which proved to be a papilloma.

Dr. CUTLER had a case similar to that of the speaker's. He first resorted to removal but the growth recurred. He then tried radium emanations which failed to prevent its spreading to the lids and lachrymal gland. He finally exenterated the orbit and there has been no recurrence up to the present time.

Dr. J. BRUDER showed for Dr. MARPLE a case of **bilateral coloboma of the optic nerve and choroid.** Remains of the hyaloid artery could be seen and there was lateral nystagmus.

Dr. L. EMERSON showed two cases in which he had successfully removed **a piece of steel from the interior of the eye.**

In the first case the foreign body passed through the lens and lodged in the vitreous. It was located by means of an X-ray and removed through a scleral opening. After an hyphæma had become absorbed, and the eye free from inflammation, a small opacity could be seen in the lens indicating the path of the steel. Up to the present time, several months since the accident, the opacity has not increased in size. Vision in the eye is $\frac{5}{200}$.

The second case is very much like the first; the path of the steel was through the cornea and lens. It was removed through a scleral opening also. The opacity in the lens now, three months since the accident, is no greater than it was at the time of the patient's recovery. He still has useful vision. Dr. Emerson then referred to a patient of his in whose right lens a foreign body was retained for two years with only a circumscribed opacity. Then in a very short time, the whole lens became opaque.

Dr. GEORGE H. BELL showed a case of **retinitis pigmentosa.** Patient, male aged 50, showed irregular and slightly contracted fields and an annular scotoma in each eye. There was an unusual amount of pigment in the equatorial region of each

fundus and a retinitic atrophy of the papillæ. The Wassermann test was negative. Vision in each eye was reduced to $\frac{20}{100}$. The patient suffered from night blindness.

There was considerable difference of opinion as to the diagnosis. Dr. CLAIBORNE thought that there was an accompanying chorioretinitis. Dr. LAMBERT thought it a case of chorioretinitis. Drs. MARPLE, CUTLER, and COHEN thought it an atypical retinitis pigmentosa. Dr. WOOTTON thought that the deposition of pigment was too profuse and that there must be some choroidal involvement.

Dr. C. BERENS asked if there was any difference in the pathology of the two diseases. Dr. BELL said that according to text-books, the pathology is practically the same. They both begin as a degeneration or atrophy of the chorio-capillaris. In retinitis pigmentosa, there is an early migration of pigment into the retina which slowly advances with the progress of the disease. In chorioretinitis the deposition of pigment is a late manifestation. The history of the case is a great aid to the diagnosis.

Dr. BELL also showed a case of **keratoiritis** with fibrinous exudate in the anterior chamber of each eye in a child of 7 years of age. When the child was first seen there was circumcorneal injection with œdema of the adjacent conjunctiva. The cornea was hazy and the anterior chamber deep. The pupil was small and the iris discolored. The fibrinous exudate extended about 2mm in height.

The child had just recovered from an attack of Grippe and at the time of entrance, her temperature was 101°. With a negative Wassermann and a negative blood culture and no sign of oral sepsis, Dr. Bell thinks the condition must have come on as a result of influenza. The eyes became normal after two weeks' treatment in the hospital.

Dr. B. W. KEY showed a case of **hyperplasia of pituitary body with Frazier's and Cushing's operation and puncture of corpus callosum, with recovery.**

The case was that of a boy of 15 years of age, who was first seen in April, 1913. His chief complaint was loss of vision in his right eye which first occurred about four months previously. He thinks there was a recovery from that attack but recently the same condition had returned. He now complains that the

temporal field of his right eye is entirely cut off. He has taken on considerable weight during the past few years and has an abnormal appetite. His father and grandfather both died of cancer. There is nothing else in his history or general condition to account for the trouble. Examination of the right eye revealed a white nerve, sharply outlined. The left optic nerve showed only a slight pallor in the temporal half. Vision in right eye reduced to fingers at one foot, in left, normal. Right field showed temporal hemianopsia; left, normal. In August, 1913, patient developed left temporal hemianopsia and an X-ray showed enlargement of the sella turcica with bony necrosis. He was referred to Dr. Chas. Frazier of Philadelphia who, after determining increased sugar tolerance and with no improvement following two weeks' treatment with organotherapy, exposed the sella by the transfrontal method, but owing to considerable oozing only a small piece of tissue which proved to be normal pituitary was removed. The wound was closed and the patient made a quick recovery from the operation but with slight weakness in the right arm and leg. The tumor was next approached intranasally by first resecting the septum up to the floor of the sphenoidal cells. A grooved director was then passed into the roof of the sphenoidal cell and held there until an X-ray showed its location which was found to be at the point desired. The following day the roof of the sinus was enlarged and opened and the floor of the sella opened and enlarged. The tumor presented in the opening. It was carefully curetted away and the wound was packed and closed without drainage. Recovery was uneventful. In March, 1914, the right eye was slightly more prominent than the left and the pupil was larger. Vision was shadows on the nasal side. The left eye showed no change in the visual field and vision was $\frac{20}{15}$. June 7th, he complained of excruciating headache lasting for several hours. August 26th, he was irritable and hysterical but free from pain. March 19, 1915. He had lost weight, grown taller and better proportioned, but his muscles were sore. There was marked muscular weakness from the result of which he had fallen recently. On May 7, 1915, patient became, suddenly, much worse. He was languid, drowsy, and mentally deficient. He had an intense headache. He was at once returned to Dr. Frazier who did a decom-

pression operation, at the same time puncturing the corpus collosum. Recovery has been so far complete. It is now one year since the last operation. Patient is free from headache. Memory and strength have returned. Vision in right eye is nil; in left, $\frac{20}{15}$, field slightly contracted, but the color fields are enlarged.

His only complaint now is a sudden dizziness on leaning forward, and a frequent dripping of clear fluid from the nose.

DISCUSSION: Dr. WEIDLER said that he had a case of **bi-temporal hemianopsia** under observation at the present time. Vision had been diminished for two years. Five months previously Dr. Elsberg decompressed the skull with temporary relief only. Both optic nerves were pale.

Dr. H. W. WOOTTON reported a case of **glaucoma following combined extraction, due to gradual ingrowth of corneal epithelium.** Patient, female aged 60 years, had had a combined extraction two years previously. For the last few months the eye had suffered from periodic attacks of pain and redness. When first seen a condition of subacute glaucoma existed. The anterior chamber was deep, the cornea was hazy but the iris could be seen. The left pillar of the iris was caught in the old scar. Iridectomy under ether relieved all symptoms for five days when pain and increased tension returned. A trephining with iridectomy was then done below which relieved the acute symptoms for a few days only. Refusing to submit to an enucleation, another trephining was done back of the ciliary body. The eye became quite soft from the escape of fluid. Within a week the eye was again of stony hardness, quite painful, and blind. It was enucleated. Microscopic sections showed that the anterior chamber was everywhere lined with epithelium Photo-micrographs illustrating the case were exhibited.

DISCUSSION: Dr. SCHIRMER asked if the diagnosis was made clinically. He said that he had seen one case due to injury. The diagnosis was not made before the eye was removed.

Dr. WOOTTON replied that he suspected the cause but needed the microscope to confirm the diagnosis.

Dr. J. GUTTMAN read a paper on **An improved method of extirpating the lacrimal sac.**

Dr. Guttman suggested the injection of the sac with methy-

lene blue in order that the wall of the sac could be more easily distinguished.

DISCUSSION: Dr. CURTIN said that Dr. Guttman's idea was not a new one. He said that Meller a great many years ago had used it. He had also injected paraffine colored with methylene blue, into the sac for the same purpose.

Dr. WOOTTON said that he did not think that it was of any value. He said that in the cases that do not bleed it is not necessary and in the cases that do bleed freely, methylene blue would be of little value. He is not a strong advocate of removing the lacrimal gland for the relief of tearing. Dr. SCHIRMER said that removing the sac did not ever relieve epiphora, except in that a source of irritation was done away with. He thinks Toti's operation better, but it should never be done where the canaliculus has been slit.

# REPORT OF THE TRANSACTIONS OF THE SECTION ON OPHTHALMOLOGY OF THE ROYAL SOCIETY OF MEDICINE.

By Mr. C. DEVEREUX MARSHALL, F.R.C.S., London.

The ordinary meeting of the Section was held on Wednesday, 14th of June, under the presidency of Mr. Priestley Smith, F.R.C.S.

Mr. G. H. Pooley sent the further notes of a case of Mikulicz's disease previously shown. A growth had been removed from the left orbit, with some difficulty. Vision in that eye steadily improved for some time, but recently there were signs of return of the disease on the same side, though the scar was free from invasion. The removed material showed the appearances of small round-celled sarcoma.

Dr. F. R. Yelland showed a patient with **loss of visual orientation,** following a wound received four months previously. Soon after being put to bed, he had a genuine epileptic seizure, and was inaccessible for fourteen hours. He afterwards had right hemiplegia, which cleared up later, but he had erroneous visual projection. The condition was now improving.

Mr. Leslie Paton, discussing the foregoing case, drew attention to the similar case under the care of Captain Smith and Colonel Gordon Holmes, reported in the *British Medical Journal*, of March, 1916. He gave the results of the tests he had carried out in the present patient. He satisfied himself that the faulty projection was not due to defective eye movements. He believed that there had been complete destruction of the right occipital cortex, the left sensory visual cortex having escaped fairly well. There seemed to have been a

complete severance of the superior longitudinal commissural fibers.

Captain CARRUTHERS exhibited a case of **retinitis pigmentosa** of unusual character, in a young man who had been a soldier three months. There was no consanguinity, but a younger brother and an older sister suffered from night-blindness, and as this patient could not see to drive when dusk came on, he declared sick, and so came under observation.

The case was discussed by Mr. W. LANG, Mr. STEPHEN MAYOU, Mr. J. B. LAWFORD, the PRESIDENT, and Mr. LESLIE PATON.

Captain A. C. HUDSON exhibited, in a tentative form, a **giant perimeter,** and invited suggestions as to improvement.

Lieut.-Colonel R. H. ELLIOT gave his experiences with a giant perimeter which he had made, and suggested possible improvements.

Dr. A. S. COBBLEDICK read a paper on **Four cases of pituitary tumor.** The cases were all in women, three over sixty years of age. The first case showed a contraction of the visual field of 10° to 20°, and a scotoma for color upwards and outwards from the central fixation point. The color scotoma bothered her a great deal when reading, and people's faces appeared bluish. There was particular contraction in the temporal half of the field. Later she had Cheyne-Stokes breathing, very violent headache, and became drowsy. The pupils, however, were equal in reaction, and there was no papillitis; the urine was normal. Post-mortem, a pituitary tumor, the size of a walnut, was found, and Dr. Buzzard regarded it as a cyst. There seemed to have been sufficient normal gland substance left to ensure normal metabolism during the six years that the symptoms lasted. The second case had right homonymous hemianopia and myxœdema. The memory had become bad, and the speech slurred and indistinct. The family and previous personal histories were good. She had well marked myxœdema, and there was a lesion of the left optic tract, probably due to pituitary growth. Thyroid extract was given, and there was improvement in the memory and in some numbness which had been present. The appearance of the disks and the constitution of the urine were normal. There was little material change in the patient's

condition for three years, but after that there was fairly rapid deterioration of vision, and the nervous condition increased: there were also vertigo, and flickerings in front of the eyes. Her feet and hands also became larger. Later the symptoms somewhat resembled those of Ménière's disease, but there was no deafness nor sickness. Case 3 had optic atrophy, obesity, myxœdema, and diabetes. During four years her sight had been gradually changing, and during the last year she had become very sleepy and drowsy. There was no tendency to hemianopia in this case. Her feet and hands were now definitely larger than twelve months ago, and the memory worse. Skiagrams showed an enlarged and lobulated sella Turcica. Operation was declined. He suggested that possibly the diabetes was due to implication of the posterior lobe of the pituitary; or it might be caused by pressure on the medulla. The fourth case was aged fifty, and her condition the author diagnosed as early optic atrophy, myxœdema, and pituitary tumor. She had defective memory, thinning of the hair, suffocating feelings in the throat, heart attacks, and twelve months ago she was so ill that her life was despaired of. Her disks appeared normal, but the field for white was contracted in every direction. Skiagrams showed distinct enlargement of the pituitary body, especially at the posterior part. He regarded this as a suitable case for operation.

The paper was discussed by the President, Mr. LESLIE PATON, Mr. STEPHEN MAYOU, Mr. WALTER JESSOP, and Mr. ARNOLD LAWSON, and the author replied.

# QUARTERLY REVIEW OF THE PROGRESS OF OPHTHALMOLOGY.

By H. KOELLNER, Berlin; W. KRAUSS, Marburg; R. KÜMMELL, Erlangen, W. LOEHLEIN, Greifswald; H. MEYER, Brandenburg; W. NICOLAI, Berlin; H. PAGENSTECHER, Strassburg; K. WESSELY, Würzburg; and M. WOLFRUM, Leipsic, with the Assistance of Drs. ALLING, New Haven; CALDERARO, Rome; CAUSÉ, Mayence; DANIS, Brussels; GILBERT, Munich; GROENHOLM, Helsingfors; v. POPPEN, Petrograd; TREUTLER, Dresden; and VISSER, Amsterdam.

Edited by MATTHIAS LANCKTON FOSTER, M.D., F.A.C.S., New Rochelle, N. Y.

## FOURTH QUARTER, 1915—*Continued.*

### XV.—IRIS AND PUPILS.

68. DESCHWEINITZ, G. E. **Progressive essential atrophy of all the layers of the iris.** *Trans. American Ophthalmological Society*, 1915.

69. NEEPER, F. R. **Carcinoma, apparently primary, arising from the ciliary processes.** *Annals of Ophthalmology*, October, 1915.

70. VAN DER SCHEER. **Adrenalin mydriasis in mental disease and health.** *Neurologisches Zentralblatt*, 1915, 17.

DESCHWEINITZ (68, **Progressive essential atrophy of all the layers of the iris**) presents the case of a young woman aged 23 whose iris of the left eye began to change color about three years ago. There was progressive thinning until several apertures appeared simulating polycoria. Later the iris almost entirely disappeared except for a few threads. There were no signs of inflammation and no other lesions of the eye. There was a possibility of latent tuberculous glandular infection and low nervous tone.

ALLING.

Primary sarcoma of the ciliary body is very rare. NEEPER (69, **Carcinoma of the ciliary processes**) describes the case of a

man seventy-one years of age in whose eye was found a tumor extending backward from the ciliary body 13*mm*. The cells were epithelial like those of the ciliary processes from which the tumor arose. There was but little pigment present.

ALLING.

The claim has been made that adrenalin produces a change in the size of the pupil, either as a mydriasis or as a myosis, in many patients with mental troubles, while it does not in normal persons, almost never in functional psychosis, and does so not seldom in organic diseases of the brain. To test this claim VAN DER SCHEER (70, **Adrenalin mydriasis in mental disease and health**) made 575 observations on 283 persons. He gives the percentages of positive, negative, and doubtful reactions obtained in various forms of mental disease and in health, and concludes that as a diagnostic aid in the differentiation of functional or organic psychoses it is of no practical value.

## XVI.—LENS.

71. FISHER, W. A. **Fifty consecutive intracapsular cataract extractions.** *Annals of Ophthalmology*, October, 1915.

72. KNAPP. **The beginning of senile cataract.** *Festschrift for the Fiftieth Anniversary of the University Eye Clinic at Basel*, p. 51.

73. LOEWENSTEIN. **Lenticular opacities caused by lightning.** *Klinische Monatsblaetter f. Augenheilkunde*, November-December, 1915, p. 592.

74. ROBINSON, WILLIAM. **Glassworker's cataract.** *Ophthalmoscope*, November 1st.

75. ZEIGLER, S. L. **Hereditary posterior polar cataract, with report of a pedigree.** *Trans. American Ophthalmological Society*, 1915.

KNAPP (72, **Beginning senile cataract**) comes to the following conclusions from an examination of 299 patients with incipient cataract. Senile cataract commonly presents the same character in both eyes at its onset, when three principal forms can be distinguished. 1. A circular beginning with points or striæ, usually radiating, in the equatorial region, which may be subcapsular or supranuclear. This form includes more than a third of all cases. 2. A beginning in the lower half of the lens, usually to the nasal side and below, most often in the form of a crescentic opacity parallel to the equator in the posterior cortex, about 1 or 2*mm* from the margin of the lens, rather less often as a large radiating wedge. This form includes over half of the cases. 3. A nuclear beginning is present

in 1.5% of the cases. 4. The cataract begins at the posterior pole of the lens in about 6.3% of the cases.

Visual results in the cases reported by FISHER (71, **Fifty consecutive intracapsular extractions**) were 36 with $\frac{15}{15}$ or better, fourteen with $\frac{15}{15}$ or lower. The causes for the cases of poor vision were variously assigned to central choroiditis, detachment of the retina, optic atrophy, congenital amblyopia, etc. One eye was completely lost from hemorrhage of the choroid. No post-operative inflammations are recorded.

ALLING.

Literature contains many examples of hereditary ocular defects, but none more striking than those of the lens. ZEIGLER (75, **Hereditary posterior polar cataract**) relates the history of a family of seventy-nine members extending over four generations in which twenty-four cases of congenital cataract appear, nearly all posterior polar. Various theories of the underlying cause are discussed.

ALLING.

ROBINSON (74, **Glassworker's cataract**) examined eighty-seven glassworkers, over thirty-seven years of age, and found that twenty-two had cataract. He reports forty cases, sixteen of which were seen in early stages, thirteen of which were posterior cortical in form, three ordinary senile forms with cortical radial opacities. According to his observation the opacity first appears at posterior pole of the lens immediately under posterior capsule. By oblique illumination it is brass colored, disk-like in form, and shades off toward the equator; it then clings closely and spreads to the posterior capsule and may become saucer shape. From this it gradually spreads to the rest of the cortex and may not be distinguishable from senile cataract. Dr. Legg's researches are also included in this paper. He examined five hundred and thirteen persons exposed to furnace glare in glass work, and two hundred and seventy-eight not so exposed, and he finds that persons exposed suffer more than ten times as frequently as those not exposed. In discussing the rays which cause cataract Robinson concludes that it is heat rays (infra-red) and advises the use of protective glasses which absorb most of these rays. This paper should be read in the original.

CURRAN.

LOEWENSTEIN (73, **Lenticular opacities caused by lightning**) had the opportunity to examine the eyes of five soldiers who had been struck by lightning and showed no signs of ocular disturbance. He found in all five, in both eyes of three, punctate, sharply defined opacities in the most posterior layer of the lens, or in the posterior capsule. No change took place in these opacities during the period of observation.

## XVII.—CHOROID.

76. BARKAN, H. **Postoperative detachment of the choroid.** *Journal A. M. A.*, October 30, 1915.

77. TOOKE, F. **Tuberculous meningitis with special reference to tubercle of the choroid and its pathological manifestations.** *Transactions of the American Ophthalmological Society*, 1915.

78. WEEKS, J. E. **Report of two cases of metastatic carcinoma of the choroid and one case of myxosarcoma of the orbit.** *Ibid.*

BARKAN (76, **Postoperative detachment of the choroid**) thinks that many cases of this sort are overlooked after cataract extraction, iridectomy, and trephining. The anterior chamber remains shallow and the eye soft. He believes that such detachments are to be found in 4% of cataract operations, 10% of trephining, and 20% of Lagrange. The anterior chamber is not shallow because of the detachment but the lesion is secondary to the condition of the chamber and tension. The fluid is probably derived from the choroidal vessels. There is little danger of permanent harm as the cases all become reattached.

ALLING.

TOOKE (77, **Tuberculous meningitis and tubercle of the choroid**) reports on 102 cases of tuberculous cerebrospinal meningitis but only 73 of them were followed by frequent and complete examinations. Pathologic findings were obtained in 58. The ages varied from one to sixty years but the majority of the cases were between two and thirty. There is no doubt but that the eye lesions appear very late in the disease, even a few hours before death. The earliest manifestation of tubercle, which is usually found at the bifurcation of a large vessel, is a dilated choroidal capillary followed by perivasculitis and ultimate strangulation of the vessel. The process never involves the sclera but extends up to the membrane of Bruch. Tubercles vary in size from 0.5*mm* to 2*mm*. ALLING.

WEEKS's (78, **Metastatic carcinoma of the choroid**) first case was a woman of sixty-four with recurrent carcinoma of the breast, who developed intraocular tumor. On account of the usually short life (six to seven months) after ocular metastases appear, the eye was not enucleated until six weeks later increased tension and pain ensued. The second case was a woman of forty-six. Enucleation was done for an intraocular tumor which proved to be carcinoma but no growth could be found in any other part of the body until after the patient's death, three months later, a carcinoma of the right lung was discovered. The third case, a child of six, presented extreme swelling of the upper lid, pushing the eye downwards. The tumor was tense and elastic. There were no glandular enlargements. The mass was removed to the apex of the orbit but there are now evidences of recurrence.

ALLING.

## XVIII.—GLAUCOMA.

79. BOEHM. **Contributions to the pathological anatomy and operative treatment of congenital hydrophthalmos.** *Klin. Monatsbl. f. Augenh.*, Nov.-Dec., p. 556.

80. HARROWER, D. **A report of several cases of chronic glaucoma treated by iridotasis.** *Transactions American Ophthalmological Society*, 1915.

81. KRAUPA. **Elliot's operation and cyclodialysis.**

HARROWER (80, **Chronic glaucoma treated by iridotasis**) dissects up the conjunctiva after an incision 10*mm* above the corneal edge. He then makes an incision in the sclera with a keratome near the cornea and grasping the free edge of the iris pulls it up into the wound, leaving it with the under surface upward. The conjunctiva is then placed in position over the scleral wound and incarceration of the iris. He reports six successful cases.

ALLING.

KRAUPA (81, **Elliot's operation and cyclodialysis**) mentions that in Teplitz he has the opportunity to observe many cases of glaucoma, for which he blames the in-breeding of the people of the place, a tendency to arteriosclerosis, and a frightful abundance of syphilis. He is losing confidence in Elliot's operation for the results he has obtained with it are no better than those obtained by means of cyclodialysis, which he prefers.

BOEHM (79, **Congenital hydrophthalmos**) reports four cases. Schlemm's canal could not be found in any of the eyes, many signs of degeneration were present, especially hyaline degeneration of the cornea, absence in places of Bowman's membrane with partial replacement by a connective tissue rich in spindle cells, lacerations of Descemet's membrane, opacities of the lens, atrophy of the retina and choroid, with colloid deposits on the latter. The papilla was excavated in all four cases. In one case the cornea was stained with blood, evidently from diffusion from the anterior chamber. The entire Descemet's membrane was overlaid with a newly formed vitreous membrane which extended over the angle of the chamber, the iris, and the ciliary processes. In the second and third cases there was a corneal ulcer, together with a similar new formation of vitreous membrane extending out from the posterior surface of the cornea. In the second case there was an anterior detachment of the choroid, most marked below. The same condition was present in the fourth eye. As such detachments usually are associated with diminution of the tension they can be regarded to a certain degree as a sort of attempt to cure. Iridectomy was performed in all four cases with no good permanent result, as shown by the eventual enucleations. Yet in two eyes in which an incarceration of the iris occurred useful vision was maintained for a fairly long time. There is no agreement as to the effects produced by the various methods of operating, or concerning the kind of operation to be chosen.

## XIX.—RETINA, OPTIC NERVE, AND VISUAL TRACT.

82. CUNY. **A rare ophthalmological condition.** *Festschrift for the Fiftieth Anniversary of the University Eye Clinic at Basel.*

83. DAVIS, A. E. **Spontaneous reattachment of the retina after twenty years' duration. Report of case with remarks.** *Trans. Amer. Ophth. Soc.*, 1915.

84. PARKER, W. R. **Sclerotrephine operation for detached retina.** *Jour. A. M. A.*, November 13, 1915.

85. PUSEY, B. **Family degeneration of the macula lutea with suggestion as to its cause.** *Transactions American Ophthalmological Society*, 1915.

86. THOMSON, E. S., and CURTIN, T. H. **Detachment of the retina.** *Journal of the A. M. A.*, Nov. 13, 1915.

CUNY (82, **Rare ophthalmological condition**) reports an unusual sort of connective-tissue formation on both papillæ

of a 13-year-old boy, which increased in size during two years of observation.

THOMSON and CURTIN (86, **Detachment of the retina**) recommend making a *2mm* to *3mm* trephine opening in the sclera as far back as possible between the external and inferior recti muscles. A flap including conjunctiva and Tenon's capsule should be carefully dissected up. There is usually little or no reaction. A week or ten days later the needle of a small aspirating syringe is inserted into the subretinal space through the scleral opening and the subretinal fluid is forcibly removed. Three cases are reported, in one of which a satisfactory reattachment seems to have taken place. It is emphasized that the procedure gives no promise of success in old cases where there are inflammatory products in the vitreous, when the tension is low or in high myopia.

ALLING.

The detachment in the case reported by DAVIS (83, **Spontaneous reattachment of the retina**) evidently took place after injury when the patient was six years old. This was eleven years before he came under observation. He was examined occasionally over a period of eleven years and then spent six years in the country greatly to the improvement of his health. After that time his retina was found to be in place but the field was not restored. The patient was tuberculous and it is suggested that there may have been choroidal lesions which in healing bound down the retina. ALLING.

PARKER (84, **Sclerotrephine operation for detached retina**) gives histories of eleven cases treated by this procedure. The vision was improved in four, unimproved in five, and made worse in two. The fields were improved in eight and made worse in three. He thinks that this treatment gives more promise than any other. ALLING.

The lesion in the cases of PUSEY (85, **Family degeneration of the macula**) was confined to an oval area of about two disk diameters about the macula and showed the mottled appearance characteristic of senile degeneration. The explanation for the occurrence of these cases is a premature arteriosclerosis which showed as a family tendency. There are records in literature of eight families showing similar lesions.

ALLING.

## XX.—ACCIDENTS, INJURIES, FOREIGN BODIES, PARASITES.

87. APPLEMAN, L. F. **Triple rupture of the choroid with iridodialysis.** *Ophthalmology*, October, 1915.

88. KRUECKMANN. **Wounds of the eye in battle.** *Zeitschrift f. Aerztl. Fortbildung*, 1915, 18.

89. LOEWENSTEIN. **Ophthalmological observations from the front row of field hospitals.** *Prager med. Wochenschrift*, p. 259.

90. PAGENSTECHER. **Injury of the retina by increased air pressure.** *Muenchener med. Wochenschrift*, p. 1586.

91. REDSLOB. **Dislocation of the globe and successful replacement.** *Klinische Monatsblaetter f. Augenheilkunde*, November–December, p. 582.

92. SCHIECK. **Wounds of the eye.** *Muench. med. Wochenschr.*, p. 1435.

93. SCHMIDTMANN. **Direct and indirect wounds of the optic nerve.** *Zeitschrift f. Augenheilkunde*, July–August, p. 77.

94. UHTHOFF. **Ophthalmological experiences and notes of the war.** *Medizinische Klinik*, p. 131.

APPLEMAN'S (87, **Triple rupture of the choroid with iridodialysis**) patient was struck in the eye with a baseball and the anterior chamber was found to be half filled with blood. After this had cleared away iridodialysis was discovered together with three vertical ruptures of the choroid, two long and two short, on the nasal side. He was able to find only six similar cases reported.

ALLING.

SCHIECK (92, **Wounds of the eye**) speaks of the injuries inflicted upon the nervous and muscular apparatus of the eye by shot wounds of the orbit, and calls attention to the fact that serious lesions may be produced at the posterior pole when the shot does not pass through the orbits at all, but through the maxillary sinuses. After a wound of the left pontine visual center by a fragment of shrapnel there appeared a paresis of the left abducens and facialis, with conjugate deviation to the right.

KRUECKMANN (88, **Wounds of the eye in battle**) says that the missiles most responsible for wounds of the eye are rifle bullets, then shrapnel, and finally grenades. The wounds that are to be considered the most dangerous are those that pass obliquely through both orbits. In addition to the direct injury the explosionlike effect of these wounds must be taken into account as the force of the projectile exerts such a pressure on

the globe as to burst it to pieces in consequence of its fluid contents, so that particles of the membranes of the eye are to be found in the orbital tissues, or even in the accessory sinuses. Less frequently bursting effects are to be observed in the bones of the orbits, and it is highly probable that these wounds are caused by missiles that come from a distance of not more than 700 to 800 meters. When they come from greater distances, holes are made, in consequence of which the eyeball usually undergoes inflammatory atrophy. When the floor of the orbit is broken up a diminution of the eye may be counterfeited by cicatricial adhesions of the eye muscles to the splinters of bone which cause the eye to be retracted. A serious feature of shot wounds of the head is the appearance of fissures of the bony walls of the orbit, either at the optic foramen or in the posterior wall of the frontal sinus, as through these a fatal meningitis may be set up if the patient should contract a sore throat or a cold in the head. He describes some cases in which Roentgenographs failed to disclose destruction of the walls of the orbit which was later discovered at the autopsy. Finally he speaks of the danger of sympathetic ophthalmia from bits of uveal tissue left behind, and of trephining in choked disk.

LOEWENSTEIN (89, **Ophthalmological observations from the front row of field hospitals**) gives some hints for the aid of the military surgeon who is not specially trained in ophthalmology. He calls attention to the danger of corneal ulcers in cases of facial paralysis and urges the application of a dressing to the eye on the injured side. Severe cases of conjunctivitis caused by the presence of foreign bodies, such as cigar ashes, are not uncommon. Advancing corneal ulcers he has often cured by painting the progressive margins with iodine tincture. When the eyeball is wrecked he prefers enucleation to exenteration if the wound has opened a direct communication between the orbit and the nose or an accessory sinus and so increased the danger of infection.

UHTHOFF (94, **Ophthalmological experiences in war**) reports his observations on 600 soldiers. Trachoma was present in only five per cent. Iritis caused by rheumatism was observed frequently. Forty-six per cent. of the wounds of the eye led to blindness. No cases of sympathetic ophthalmia were observed and none of inferior hemianopsia. Simulation was present in

only one-half of one per cent. of the cases. The ratio of bilateral to unilateral blindness is 1:10.

PAGENSTECHER (90, **Injury to the retina by increased air pressure**) describes two cases of injury of the retina by the increase of air pressure caused by grenades exploding in the near vicinity. The region of the macula was clouded and the foveal reflex was blotted out, so he thinks that a delicate exudation takes place in the region of the macula in these cases.

SCHMIDTMANN (93, **Direct and indirect wounds of the optic nerve**) reports a case in which the patient was hooked by the horn of a cow in such a way that a double lesion was produced, a partial laceration of the eyeball, and a partial severance of the optic nerve in the optic canal through a fissuring of the roof of the orbit.

In the case described by REDSLOB (91, **Dislocation of the globe and successful replacement**) the eye was displaced into the maxillary sinus by the horn of a cow, all the recti and the superior oblique being torn. The eyeball was replaced in the orbit and secured by sutures in the conjunctiva and sclera, where it became reattached in position, though with extremely limited movements. Optic atrophy supervened in consequence of a laceration of the optic nerve, and the eye also presented ruptures of the choroid in the region of the macula.

VII.—**Principles and Practice of Perimetry.** By Dr. LUTHER C. PETER, Philadelphia. Pages 224, with 119 illustrations. Published by Lea & Febiger. Price, $2.50 net.

The object of Dr. Peter in presenting this work is twofold: First, to give to the student in ophthalmology and ophthalmo-neurology a collective study of the perimeter and its application, and, second, to stimulate a greater interest in the practice of perimetry. Ophthalmic literature has long needed a work of this nature as the only works dealing exclusively with the subject are in German. An introductory chapter gives the general considerations and normal field. The color fields are more nearly those found by the writer, than the fields usually given. They still seem a bit too large, especially those for blue and red. The chapter on methods explains fully those which the author has found most useful. His preference is for his hand campimeter, which is a miniature of the campimeter of von Graefe, 14 inches square, and is fitted with a cheek rest and handle. The tests are made in the same way as they are on the large campimeter, Bjerrum's screen, or Duane's tangent curtain. Its advantages are especially portability and uniform illumination, and the author claims greater accuracy in results. The other methods are described less fully but give a working knowledge of the same. The anatomy of the visual tract is covered concisely as are the general pathological conditions. In the chapter devoted to special pathological fields, the author has collected in 100 pages the fields as found in the various diseases. The fields in the diseases of the chiasm are especially worthy of study as they show the fields before and after treatment. The work is concise, practical, well written, and with the complete bibliography is a most convenient book for reference or study.

J. W. WHITE.

VIII.—**Methods of Refraction.** By Dr. JAMES THORINGTON. Pages 407, illustrations 344. Published by P. Blakiston's Sons & Co., Philadelphia, 1916. Price, $2.50 net.

The author's previous books, *Refraction and How to Refract*, *Prisms* and *Retinoscopy*, are now collected in one volume which has required some rearrangement of the subject matter. The new book is particularly intended for beginners and the required mathematical knowledge is not exacting. It imparts a great deal of information, treating optics in general, optics of the eye in particular, the use of the ophthalmoscope, the various refractive conditions, retinoscopy, muscles, applied refraction, etc., and includes a chapter on retinoscopy without a cycloplegic combined with the fogging method. The collection of these various subjects in one volume will be a convenience to the reader and it should continue to be one of the popular works on refraction.

## OBITUARY.

NEW YORK, Oct. 18, 1916.

The Board of Surgeons of the New York Eye and Ear Infirmary record with deep sorrow and sincere regret the death of the late Dr. WILBUR BOILEAU MARPLE, who died suddenly at Kennebunkport, Maine, on September 30, 1916.

Dr. Marple had been connected with the Infirmary for twenty-five years, first as Assistant Surgeon, and from 1901 to the time of his death, as Attending Surgeon. He was also for a number of years one of the representatives of the Board of Surgeons on the Board of Directors.

The death of Dr. Marple has removed from among us a distinguished Ophthalmologist, admired colleague, wise counsellor, and honored friend—his loss is greatly deplored by all of us. The Board of Surgeons desire to express their appreciation of his high professional attainments, and extend to his bereaved family their profound sympathy.

(Signed) JOHN E. WEEKS, M.D.,
EDWARD B. DENCH, M.D.,
W. E. LAMBERT, M.D.

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# School of Ophthalmology

**Instruction in Ophthalmology is given at the HERMAN KNAPP MEMORIAL EYE HOSPITAL, 57th Street and 10th Avenue, in (*a*) a systematic course covering all branches, beginning October and February; and in (*b*) short practical courses on External Diseases, Refraction, Ophthalmoscopy, Operative Surgery, Pathology, and Physiological Optics. For particulars apply to Dr. Arnold Knapp, at the Hospital, 500 West 57th Street, New York City.**